A Dissertation on

"COMPARISON OF ALLEN STROKE SCORE AND GREEK STROKE SCORE WITH CT BRAIN IN CLINICAL DIAGNOSIS OF ACUTE STROKE"

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CERTIFICATE

Certified that this dissertation in **"COMPARISON OF ALLEN STROKE SCORE AND GREEK STROKE SCORE WITH CT BRAIN IN CLINICAL DIAGNOSIS OF ACUTE STROKE"** is the bonafide dissertation done by **Dr.S.MENAKA** and submitted in partial fulfilment of the requirements for the Degree of **M.D. General Medicine** Branch I during the academic year 2016-2019 of **The Tamilnadu Dr.M.G.R. Medical University, Chennai.**

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I solemnly declare that the dissertation titled "**COMPARISON OF ALLEN STROKE SCORE AND GREEK STROKE SCORE WITH CT BRAIN IN CLINICAL DIAGNOSIS OF ACUTE STROKE"** was done by me from JULY 2017 to JUNE 2018 under the guidance and supervision of Professor **Dr. KUMAR NATARAJAN M.D.,**

This dissertation is submitted to **The Tamilnadu Dr.M.G.R Medical University** towards the partial fulfilment of the requirement for the award of MD Degree in General Medicine (Branch I).

Place: Coimbatore **Dr.S.MENAKA**

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The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation Proposal is accepted and you are permitted to proceed with the above Study.

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INTRODUCTION[1]

Stroke is known to physicians since the time of Hippocrates, since 2400 years ago. At that time, stroke is called as 'Apoplexy' which means 'struck by violence'. The cause for apoplexy was in search to many physicians. In middle of 1600, Jacob Wepfer discovered that occlusion of blood vessel can cause apoplexy. In 1928, it was named as Cerebral Vascular Accident (CVA).The term accident denotes the emergent call for action. Most stroke victims are having good chance of recovery only when early intervention is started[24]. The stroke scoring systems are designed for hospital screening, to cut-short the time needed to diagnose the cause for stroke. Early thrombolytic therapy can be started after ruling out the intra-cerebral haemorrhage. The scoring systems like Allen score, Greek score, Siriraj score and JUST (Japan Urgent Stroke Triage) score have been devised clinically, which are used all over the world to differentiate ischemic and haemorrhagic stroke where brain imaging facilities are not available.

AIMS OF THE STUDY

1) To assess the types of stroke using the clinical scoring system.

2) To determine the accuracy of stroke scores by comparing with the findings of Computed Tomography scan.

REVIEW OF LITERATURE

Stroke produces central neurological deficits of acute or sub-acute onset due to focal vascular causes. Ischemic stroke accounts for 85% of total. Haemorrhagic stroke constitutes 15% of stroke.[2] The incidence of stroke increases with age. women are less frequently affected than men up to 80 years and after 80 years ,both sex are equally affected.[2] Stroke is caused by impaired cerebral perfusion due to vascular causes like blockage of arteries by emboli, macro-angiopathy or micro-angiopathy.[6] Course of stroke may be Transient ischemic attack; Reversible ischemic neurological deficits, progressive stroke or completed stroke, type of infarction may be lacunar, border-zone, lacunar, watershed or territorial infarct.[3] The single affected vessel resulting in a specific vascular syndrome. Depending on the extent of tissue damage caused by impaired blood supply determines the recovery of neurological deficits.

BLOOD SUPPLY OF BRAIN:[6]

The brain uses 20% of available oxygen for normal function, making tight regulation of blood flow and oxygen delivery critical for survival. Auto-regulation of cerebral blood flow is the ability of the brain to maintain relatively constant blood flow despite changes in perfusion pressure.[6] Auto-regulation is present in many vascular beds but well developed in the brain. In normotensive adults, cerebral blood flow is maintained at 50ml per 100gram of brain tissue per minute, provided CPP is at the range of 60-160mmHg. Above

and below this limit, autoregulation is lost and cerebral blood flow becomes dependent on mean arterial blood pressure. When CPP falls below the lower limit of auto-regulation, cerebral ischemia occurs.

The longer the cerebral ischemia lasted the normal function is less likely to be regained. The zone of tissue which the local cerebral perfusion lies between the functional threshold and the infarction threshold is called the ischemic penumbra.[4] Within the penumbra, perfusion is reduced, but diffusion is normal. If the occluded vessel is re-canalized earlier, the tissues in the penumbra can largely survive and regain its normal function. The penumbra thus represents the tissue at risk for further stroke, which can be salvageable by revascularisation techniques. Hence imaging the penumbra at the earliest is important for clinical decision making.[4]

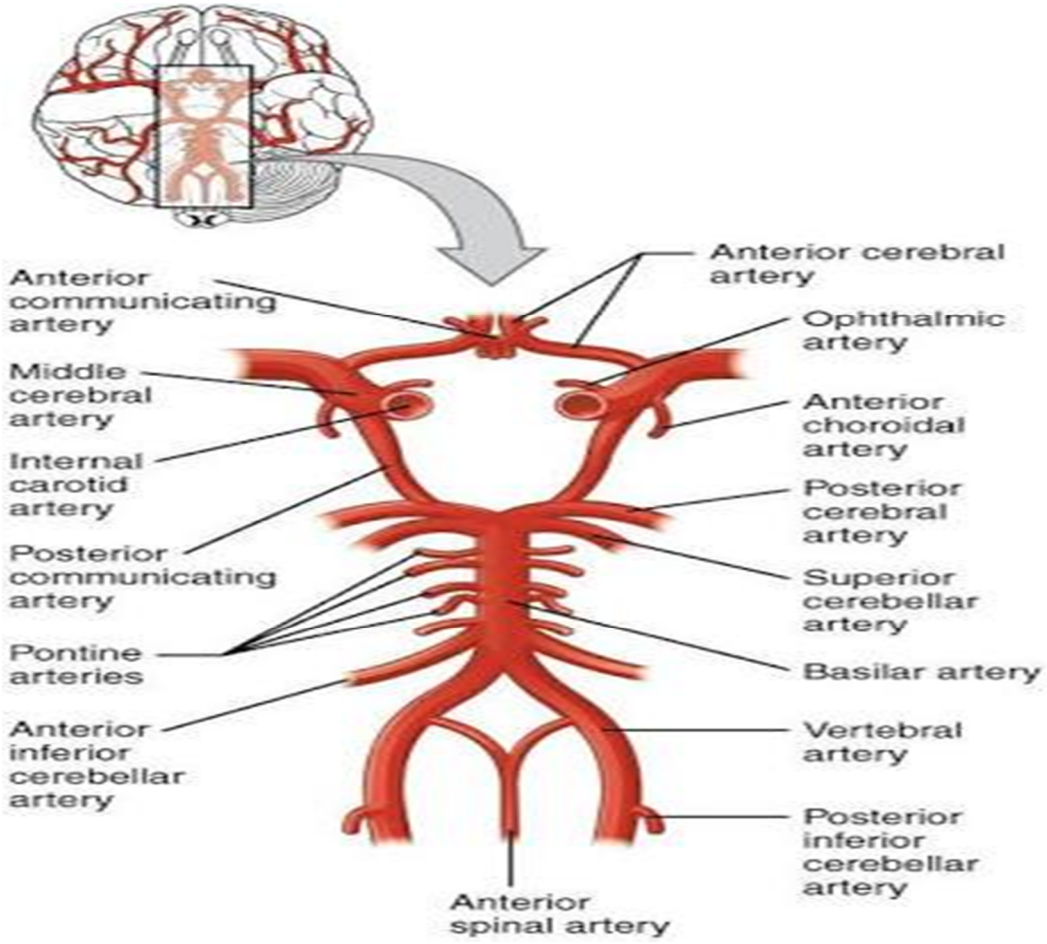
ANATOMY OF CEREBRAL CIRCULATION:[5]

The blood supply of brain is dependent on circle of Willis, which is named after an English physician Dr.Thomas Willis. Circle of Willis is a circulatory anastomosis that supplies blood to the brain and other surrounding structures. Blood vessels supplying the brain are classified into anterior and posterior.

The anterior circulation consists of internal carotid artery and its branches.

The posterior circulation consists of basilar artery and its branches.

Internal carotid artery is one of the two terminal branches of common carotid artery.



The internal carotid artery is divided into seven segments according to Bouthillier nomenclature. The segments of the internal carotid artery are cervical segment, petrous segment, Lacerum segment, Cavernous segment, Clinoid segment, ophthalmic segment and communicating segment. The terminal branches of internal carotid artery are middle cerebral artery and anterior cerebral artery. The middle cerebral artery[7] continues as an extension of

Internal carotid artery, it passes laterally between the upper surface of the

Temporal lobe and the inferior surface of the frontal lobe in the Sylvain fissure to reach the surface of the insula, where it divides into four segments.[5,7]

1] M1 segment:

The sphenoidal segment, M1 named due to its origin and it tracks the sphenoid bone. This segment perforates the brain with numerous anterolateral and central (lateral lenticulo-striate) arteries, which supplies the basal ganglia.

The branches are

- Medial lenticulostriate penetrating arteries
- Lateral lenticulostriate penetrating arteries
- Anterior temporal artery
- Polar temporal artery
- Uncal artery

2] M2 segment:

It is also known as insular segment, it continues from the bifurcation to the insula, makes a bend to continues as M3.

It divides into superior and inferior trunks. The superior terminal branches are further divided into lateral frontobasal artery, prefrontal sulcal artery,

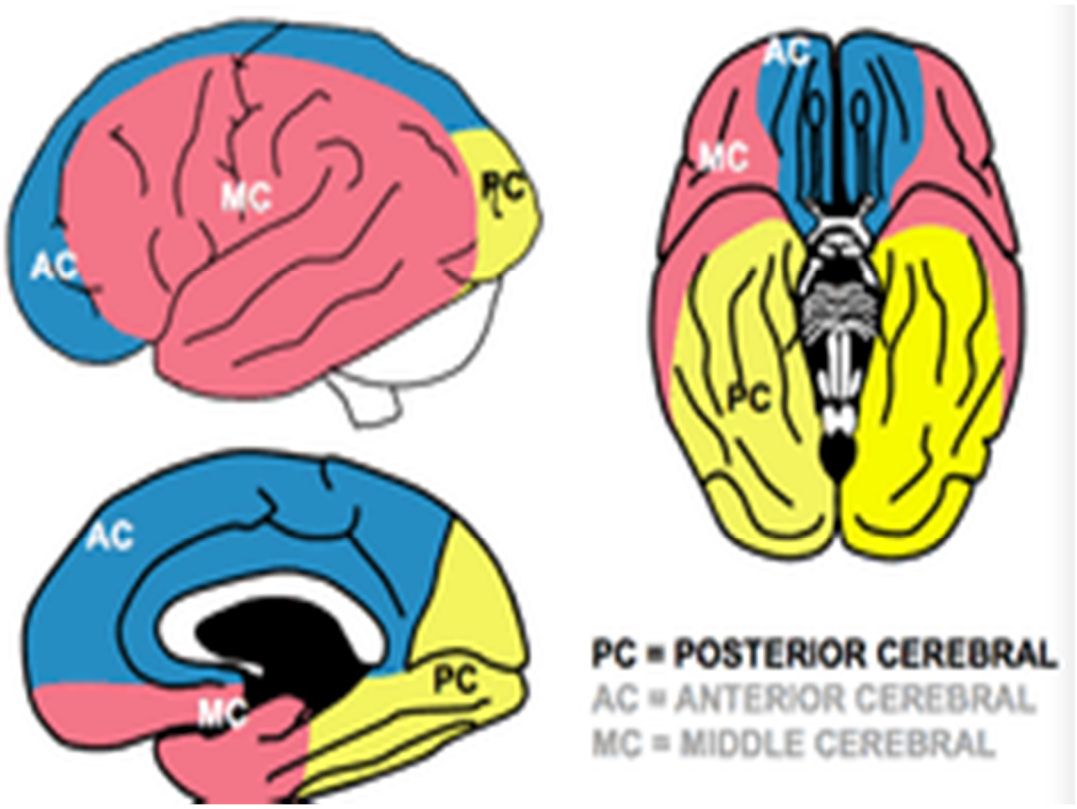
prerolandic and rolandic arteries. The inferior terminal branches id divided into three temporal and two parietal branches.

3] M3 segment:

Opercular branches arises within the sylvian fissure, it is called opercular segment.

4] M4 segment:

The branches emerges from the sylvian fissure, continues to the lateral surface of cerebral hemisphere. It is also called as cortical segment.



ANTERIOR CEREBRAL ARTERY:

Anterior cerebral artery is one of the terminal branches of the internal carotid artery.it runs forwards and medially superior to the optic nerves and enters the longitudinal fissure of the cerebrum, where it come close contact with the opposite anterior cerebral artery. Both side arteries are connected by a short anterior communicating artery. Then it passes backwards over the genu of corpus callosum to the parieto-occipital sulcus where it anastomoses with the branches of posterior cerebral artery. The branches of anterior cerebral artery are anterior communicating artery, perforating branches, medial striate artery, pericallosal artery and cortical branches.it supplies the whole of the medial surface of the cerebral hemisphere upto parieto occipital sulcus and a strip of 2.5 cm width adjoining superior aspect of the lateral surface of brain.

POSTERIOR CIRCULATION:

It is also called vertebro basilar system. Vertebral artery is a branch from first part of the subclavian artery. Due course of the artery is divided into four parts. First part extends from the origin to the entry into the foramen of transverse process of 5^{th} or 6^{th} cervical vertebra. Second part ascends through the foramina of transverse process of the c6 to c2 vertebra. Then it passes through the foramen magnum, pierces the duramater and ascends over the ventral surface of medulla to reach the lower border of pons where it joins with the vertebral artery of opposite side to form the basilar artery.

The branches of vertebral arteries are

1) Meningeal branches – supply the Dura of posterior cranial fossa and falx cerebelli.

2) Anterior spinal artery – supplies the full length of the cord.

3) Posterior spinal artery – May arises from posterior inferior cerebellar artery.

4) Medullary arteries – supplies the upper medulla.

5) Posterior inferior cerebellar artery – largest branch of vertebral artery, it supplies dorsolateral portion of medulla, choroid plexus of 4^{th} ventricle and cerebellum.

Basilar artery is formed by union of two vertebral arteries. It ascends from Ponto-medullary junction to the upper border of pons where it divides into two posterior cerebral arteries.

The branches of basilar artery are

1) Anterior inferior cerebellar artery – supplies the anterior & inferior surface of cerebellum.

2) Labyrinthine artery – it accompanies the facial & vestibule-cochlear nerve into the internal acoustic meatus, supplies the inner ear.

3) Superior cerebellar artery – it supplies the superior surface of cerebellum, pineal gland, superior and middle cerebellar peduncle.

4) Posterior cerebral arteries;

These are two terminal branches of basilar artery. It is joined by posterior communicating branch of the internal carotid artery.

The artery is divided into precommunal and postcommunal segments, the portions proximal and distal to the attachment of the posterior communicating artery. The vessel terminates as the calcarine artery supplying the visual cortex with the exception of the macular cortex at the tip of occipital pole.

PRECOMMUNAL SEGMENT BRANCHES:

- Perforating branches
- Posterior thalmo-subthalamo-paramedian artery {artery of percheron }
- Thalamo geniculate branches
- Medial posterior choroidal artery
- Lateral posterior choroidal artery

POSTCOMMUNAL SEGMENT BRANCHES:

- Short circumferential branches
- Cortical branches

CLASSIFICATION OF STROKE:

Classification of subtype of ischemic stroke:

In TOAST study [Trial of Org 10172 in Acute Stroke Treatment], [8] the neurologists classified the stroke into five sub types.

- Large artery atherosclerosis – 20%
- Cardio-embolism – 20%
- Small vessel occlusion -20- 30%
- Stroke of other determined aetiology - 5%
- Stroke of undetermined aetiology – 30-40%

Revised ASCO classification:

In the ASCO classification,[9] the stroke subtype are categorised based on the phenotyping of ischemic stroke.

- Atherosclerosis
- Small vessel disease
- Cardio-embolism
- Other causes
- Dissection

Haemorrhagic stroke [10]

- Intracerebral haemorrhage
- Subarachnoid haemorrhage

Causes of Intra-cerebral haemorrhage:[10]

In Aguilar et al study, Intra-cerebral haemorrhage is classified by its location within the brain

- Deep ICH :

  Basal ganglia & internal capsule – 35- 70%

  Brain stem - 5- 10 %

  Cerebellum - 5- 10 %

- Lobar ICH - 15- 30 %

In European study, they have proposed an aetiological classification of Intra-cerebral haemorrhage – [SMASH –U] classification[10]

- Structural lesion – 50%
- Medications- anticoagulants
- Amyloid Angiopathy – 20%
- Systemic and other cause
- Hypertension
- Undetermined

ETIOLOGY OF STROKE: [4]

1) Atherosclerosis

2) Cardiogenic embolism

   Mural thrombus due to myocardial infarction, cardiomyopathy, aneurysm and due to Rheumatic valvular heart disease and arrhythmias.

3) Venous and venous sinus thrombosis

   Septic sinus thrombosis, coagulopathy, pregnancy, drugs [oral contraceptives, glucocorticoids]

4) Hematologic disorders

   Thrombophilia due to protein C, protein S or anti thrombin-iii deficiency.

   Hemoglobinopathy - sickle cell anaemia, thalassemia.

   Hyperviscosity syndrome due to thrombocytosis, leucocytosis myeloproliferative disorders.

5) Vasculitis

   - Primary CNS vasculitis
   - Systemic vasculitis with CNS involvement e.g giant cell arteritis,
   - Takayasu arteritis, Wegener's granulomatosis
   - Connective tissue disorders- systemic lupus erythematosis,
   - Behcet disease etc.

- Infectious vasculitis – HIV, Tuberculosis, Neurosyphilis,CMV infection.

6) Toxins

- Substance abuse – cocaine, amphetamines, LSD, heroin
- Medications – sympathomimetics, ergotamines, triptans,
- Intra-venous immunoglobulin's.

7) Nonatherogenic vascular diseases

- Arteriovenous malformations
- Dissection of intracranial and extra-cranial arteries due to trauma, Marfans syndrome.
- Vasospasm after subarachnoid haemorrhage
- Hereditary vascular syndromes
- Amyloid angiopathy

8) Iatrogenic stroke

- Angiography and surgery in carotid arteries, aorta & heart
- Injection of steroid crystals, fat embolism
- Following liposuction therapy.

9) Other causes

- Vasospasm e.g in migraine
- Metabolic diseases like homocystinuria, fabry disease, MELAS etc.
- Fat & air emboli, tumour emboli, cholesterol emboli, distal emboli from giant aneurysms.

RISK FACTORS FOR STROKE: [12,13]

The effective way to reduce the stroke burden in the population involves the modification and treatment of vascular risk factors.

Modifiable risk factors:

- **Hypertension:**[14]

  Philip et al study showed that hypertension as the major risk factor for stroke with incidence of stroke is directly proportional to the level of blood pressure. The Framingham Heart study found that for every 20 mmHg systolic or 10 mmHg diastolic increases in blood pressure there is a doubling of mortality from both ischemic heart disease and stroke. The authors suggest a 10 mmHg reduction in systolic or a 5mmHg reduction in diastolic blood pressure would result in a 40% lower risk of stroke deaths.

- **Smoking:**

The nicotine and carbon monoxide in cigarette smoke damages the heart and blood vessels, it paves the way the stroke to occur[8].smoking increases blood viscosity, fibrinogen and platelet aggregation and decreases the HDL cholesterol, directly damages the endothelium and increases the blood pressure. The incidence of stroke in smokers has a relative risk 1.92 times higher than non-smokers.

- **Diabetes mellitus:[8]**

  Many people with diabetes also have dyslipidaemia and hypertension , increases the risk of stroke.

- **Diet :[8]**

  Diets rich in saturated fatty acids, trans-fat and cholesterol, modifies the lipid metabolism in the body. Diets rich in sodium can increase the blood pressure. Diet with high calories will lead to obesity. A diet containing five or more servings of fruits and vegetables per day may reduce the risk of stroke.

- **Physical inactivity:**

  Sedentary life style is associated with increase in incidence of stroke. Physical activity reduces the blood pressure, body weight, plasma fibrinogen, platelet activity and increases the tissue plasminogen activator and HDL.

- **Metabolic syndrome:**

  Ischemic stroke is significantly associated with high triglyceride level and low HDL cholesterol levels.

- **Heart diseases:**[14]

  Heart disease is the second most common cause for acute cerebrovascular accidents and is diagnosed in one third of patients with stroke. Cardio-embolic stroke is the common subtype of stroke associated with heart diseases. Atrial fibrillation and atrial flutter are the most important risk factor for the development of stroke.

  The risk of stroke is 3-4 times higher in the absence of valvular heart disease in patients with lone atrial fibrillation. The other sources of embolism are from dilated cardiomyopathy, valvular heart disease, left ventricular hypertrophy, atrial myxoma, congenital heart disease and acute coronary syndrome.

- **Alcohol**

  Chronic alcohol consumption of more than 60 mg per day is associated with increased incidence of stroke.

- **Substance abuse**

  Substances like cocaine, heroin,amphetamine causes stroke through different mechanisms like increases platelet aggregation and blood viscosity.

- **Sleep related breathing disorders:**

  Sleep apnoea causes accelerated atherogenesis, hypercoagulability, reduction in cerebral blood flow, alteration in cerebral blood flow. Sleep apnoea is the important risk factor for cerebrovascular events which is usually underdiagnosed.

- **Peripheral artery disease**

  History of intermittent claudication and thrombo angitis obliterens denotes the presence of atherosclerosis of all the blood vessels. Hence the patient will be with high risk for stroke of atherosclerotic aetiology.

- **Post-menopausal hormone therapy:**

- Oral contraceptive drugs.

  Women older than 35 years taking oral contraceptive pills daily are at high risk for stroke if they are having additional risk factors like hypertension, diabetes, migraine, history of smoking and history of thromboembolism.

- **Asymptomatic carotid stenosis:**[16,22]

  Approximately about 5% of men and 10% of women over 65 years had >50% and > 80% of carotid intimal wall thickening, who are asymptomatic, they may develop stroke at any point of time. Asymptomatic carotid stenosis has been identified as risk factor for

stroke. In cases of symptomatic carotid artery stenosis 50 – 60 %, carotid endarterectomy [CEA] or carotid artery stenting [CAS] are beneficial and reduces the risk of stroke.[21]

- Psycho social stress

Non modifiable risk factors:

- Age

  Age is a continuous risk factor for occurrence of stroke and dementia, with a two fold increase in the incidence and prevalence rates for each successive 5 years after the age of 65 years.

- Race/ethnicity

  Black patients has higher incidence of stroke than white people. Intracranial atherosclerotic disease is more common in Asian patients.

- Gender

  Men show a higher incidence of cerebral vascular disease than women.

- Family history of stroke

  Family history of stroke in a first degree relative also increases the likelihood of incidence of stroke.

- Low birth weight

Birth weight is inversely proportional to the incidence of stroke. The underlying mechanisms of this association are poorly understood but maybe related with genetic and nutritional factors.

- Prior stroke or TIA, coronary artery disease
- Genetic disorders:

The genetic aetiology of stroke is polygenic inheritance.it is more related to genetic polymorphisms influencing the well documented risk factors like diabetes , hypertension, dyslipidaemia, obesity and cardiomyopathy.

Rare monogenic disorders can cause stroke such as cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL), cerebral autosomal recessive arteriopathy with subcortical infarcts and leukoencephalopathy (CARASIL), cerebral amyloid angiopathy, moya moya disease, fabry disease, Ehlers danlos syndrome, Sneddon syndrome,

Marfans syndrome and MELAS (mitochondrial encephalopathy, lactic acidosis and stroke like episodes).

Clinical features of embolic stroke: [4]

In more than half of patients, paralysis is preceded by minor signs or one or more transient attacks of neurologic deficit [TIA].In carotid or middle cerebral artery disease, the symptoms are mono-ocular blindness, hemiplegia,

hemi-anaesthesia, speech or language disturbances. In vertebra-basilar system, the prodromal symptoms are vertigo, diplopia, numbness and dysarthria. The episode of symptoms evolves over a few hours or less. The more characteristic of athero-thrombotic stroke is the occurrence of stroke during sleep and the patient awakens with the paralysis. The headache is less severe than that of intra-cerebral haemorrhage. There is no neck stiffness.

Clinical features of haemorrhagic stroke: [19,23]

The onset of symptoms in haemorrhagic stroke is more dramatic. The symptoms may evolve gradually over minutes to hours, depending on the size of ruptured artery and the speed of bleeding. Headache, vomiting, acute hypertension and nuchal rigidity with focal neurologic deficit are the cardinal features of intra-cerebral haemorrhage. The stroke occurs while the patient is up and active. Further expansion of hematoma may cause worsening of symptoms.

Classification of cerebral ischemia: [4]

Designation	Deficits
TIA	Transient focal neurologic deficit. duration is 2-15min.
Stroke in evolution, progressive stroke	Neurologic deficits that worsens for hours or days after onset.
Completed stroke	Neurologic deficit is fully established that is irreversible or partially reversible.

Subtype of Infarct: [24]

Infarct is classified based on size of the vessel involved. They are

- Territorial infarction:

  It is due to occlusion of main trunk or major branches of cerebral arteries. The infarct may include both cortex subcortical white matter and basal ganglia.

- Watershed infarction:

  It is due to impaired perfusion in the vulnerable areas at the border between the territories of major blood vessels. Infarcts are due to macroangiopathy.

- Lacunar infarction:

  The infarcts are multiple which less than 1.5cm is. Lacunar infarcts are caused by microangiopathy. Fisher has discovered the subtypes depending on the site of lesion i.e.

  - Pure motor hemiplegia
  - Pure sensory stroke
  - Clumsy hand- dysarthria syndrome
  - Ipsilateral hemiparesis- ataxia

CLINICAL DIAGNOSIS OF STROKE

History and thorough clinical assessment is essential for diagnosis.

DIAGNOSIS OF STROKE:

IMAGING STUDIES: [18]

The imaging studies are the most reliable investigation to diagnose infarct and haemorrhage in the case of stroke to start the appropriate therapy. In the recent days, the novel techniques are available for the clinicians to define the anatomy and physiology of brain and blood vessels.

The main goals of imaging[18] are

- To identify intracranial haemorrhage
- To identify the extent of the ischemic damage to the tissue and to differentiate the infarct core and the salvageable ischemic penumbra.
- To find the anatomy of blood vessel supplying the infarct or haemorrhagic region.

1. CT BRAIN: [17]

Non contrast CT brain of the brain remains the main stay of imaging of an acute stroke. Whether the tissue is supplied by end arteries or has collateral supply influences the development of cytotoxic oedema. The changes in deep grey matter nuclei can be visible within 1 hour of occlusion up to 60% of patients.

The MCA territory infarct can be visible within 60- 70% of patients in the first 6 hours.

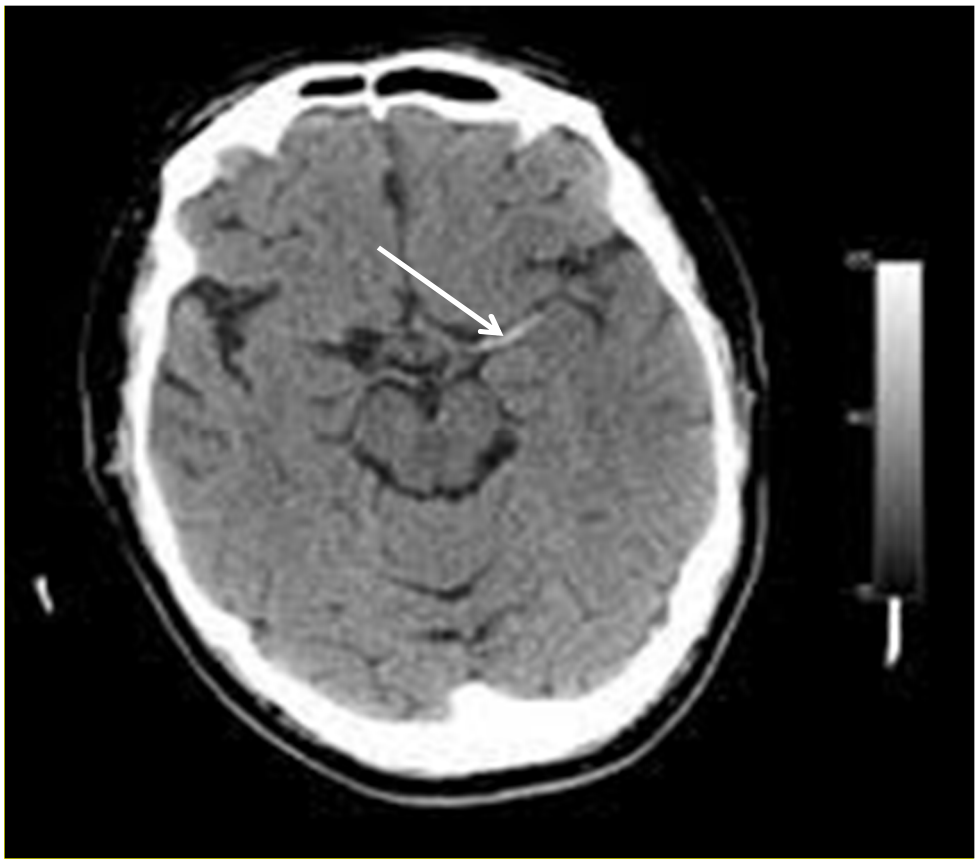
The findings in the CT brain divided into 5 phases depending on the duration of ischemia.[17]

- Immediate phase: : 0-6 hours

  The earliest CT finding is a hyper-dense segment of a vessel with direct visualisation of intravascular thrombus/embolus which is called as hyper-dense middle cerebral artery sign or middle cerebral artery dot sign.

- Early hyper-acute phase: 6- 24 hours

  The early features are loss of grey matter differentiation and hypo-attenuation of deep nuclei and Insular ribbon sign.

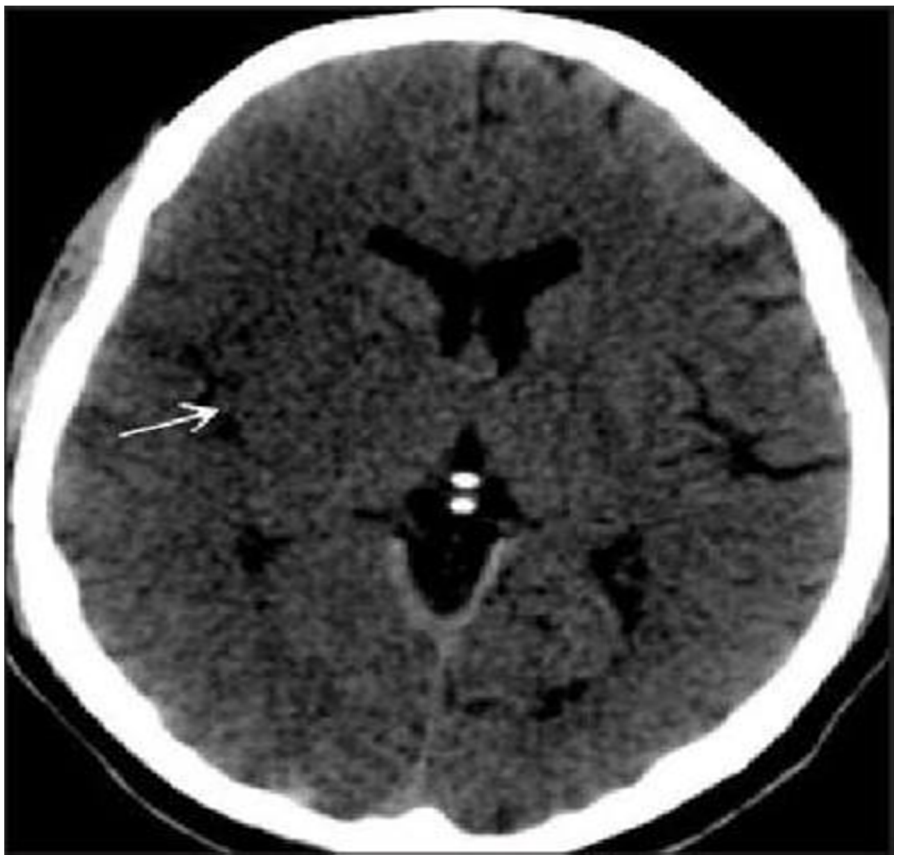


The above image showing Left MCA dot sign

- Acute: 24 hours to 1 week. As the time increases, hypo-attenuation and swelling become more marked resulting in mass effect.

- Sub-acute: 1 to 3 weeks

  Then the swelling starts to subside and small amount of petechial haemorrhages leads to elevation in the attenuated cortex. This is called CT fogging phenomenon.



- Chronic: more than 3 weeks.

  The residual swelling passes and gliosis developed which appears as with low density.

The image showing the massive infarct in the right middle cerebral artery territory.

The above image showing haemorrhage in left capsulo-ganglionic region.

2. MRI BRAIN: [20,22]

MRI imaging includes Diffusion weighted imaging, Perfusion weighted imaging and MR angiography.

MRI including the magnetic resonance angiography provides the adequate details about the diseased region either the infarct or haemorrhage with the corresponding vascular territory. Diffusion weighted imaging (DWI) shows information about the status of the tissue within the minutes of on-going ischemia.

3. ULTRASOUND METHODS: [22, 23]

- Carotid and vertebral duplex

  Carotid and vertebral artery imaging helps to intimal wall thickening and intraluminal thrombus

- Trans-cranial Doppler

- Combined duplex and TCD

ACUTE MANAGEMENT OF STROKE: [23]

The goal for management of stroke patients is to stabilise the patient and complete the initial evaluation and assessment of the patient. Critical decisions are to be made like need for intubation, blood pressure control and determination of risk/benefit for thrombolytic interventions.

According to National Institute of Neurological Disorders and Stroke, the recommended stroke evaluation time benchmarks for thrombolysis candidates.[23]

TIME INTERVAL	TIME TARGET
Door to doctor	10 min
Access to neurologic expertise	15 min
Door to CT scan completion	25 min
Door to CT scan interpretation	45 min
Door to treatment	60 min
Admission to stroke unit	3 hours

Hypoglycaemia and hyperglycaemia need to be identified and treated early in the evaluation. Both can produce symptoms that mimic ischemic stroke, but they can also aggravate on-going neuronal ischemia. Hypoglycaemia is treated with dextrose. Hyperglycaemia is treated with inj. Insulin if blood glucose is more than 200mg/dl. Adequate blood pressure target is to be achieved according to recent guidelines. Oxygen supplementation is needed if saturation is less than 94% in room air. Hyperthermia is to be avoided as it can worsen neuronal damage. Steps should be taken to prevent aspiration of food and water in the form of Ryle's tube feeding.

Treatment of Ischemic stroke: [24]

The recent treatment advances for Ischemic stroke are intravenous tissue plasminogen activator [t-PA], endovascular mechanical recanalization and intra-arterial thrombolysis.[20]

According to American stroke association guidelines for the early management of patients with ischemic stroke recommends the patients to be treated with intravenous t-PA [tissue type plasminogen activator] even if endovascular treatments are available.[19] The presence of dense MCA sign and infarct involving more than two third of MCA territory or midline shift of 5mm or more are the early predictors for neurological deterioration and mortality.

Heparin or heparinoids is useful when stroke is in progression.

Antiplatelet drugs are useful in preventing thrombotic or embolic strokes.

Treatment of Haemorrhagic stroke: [23, 24]

The treatment of acute intracerebral haemorrhage depends on the cause and the severity of bleeding. Endotracheal intubation is needed for patients with low GCS. Measures should be taken to reduce the intracranial pressure, blood pressure. The intracranial pressure is reduced by means of osmotic diuretics and by hyperventilation. In INTERECT [23][Intensive Blood pressure

reduction in Acute Cerebral Haemorrhage trial 1 & 2] study suggested that the intense reduction of blood pressure lessens the extension of hematoma.

The target blood pressure to be achieved in case of haemorrhagic stroke is 140 mmHg systolic and 80 mmHg diastolic blood pressures. Prophylactic anticonvulsant therapy should be started in all cases of haemorrhagic stroke.[22.23]

STROKE SCORES

Stroke scoring system are made for early diagnosis of stroke and the treatment can be started at the earliest to save the tissue damage and to reduce the mortality and morbidity. The available scoring systems are Allen stroke score, Greek stroke score and Siriraj stroke score.

ALLEN STROKE SCORE [28]

Allen score is also called Guy's score; it was devised by C.M.C.Allen at Guy's hospital London. It was devised to assist the physicians who are without easy access to CT scan facilities for clinical diagnosis of stroke.

Allen score is calculated based on clinical examination and history of the patient. The onset of symptoms like vomiting, neck stiffness, headache, loss of consciousness, conscious level at 24 hours, diastolic blood pressure after 24 hours of admission and history of Hypertension, Diabetes mellitus, angina and

intermittent claudication are included. Previous history of TIA or stroke and heart disease is also included in computation of score.

Fallacies of Allen score:

1. Prompt and detailed history taking is needed and clinical examination to be done completely.

2. Allen score includes calculation of clinical parameters like conscious level and diastolic blood pressure after 24 hours of admission, Hence the score could not be calculated at the time of admission.

The inference of stroke score is based on the below range;

Less than or equal to 4 denotes infarct

5-24 denotes equivocal

>24 denotes haemorrhage

Variables	Clinical features	Score
1) Apoplectic onset • loss of consciousness • headache <2 hours • vomiting • neck stiffness	none or one two or more	0 +21.9
2) Level of consciousness [24 hours after admission]	alert drowsy unconscious	0 +7.3 +14.6
3) Planter response	both flexors/ single extensors both extensor	0 +7.1
4) Diastolic BP	-	+[ ]*0.17]
5) Atheroma markers • diabetes • angina • intermittent claudication	none one or more	0 -3.7
6) Hypertension	none present	0 -4.1
7) Previous event (TIA/stroke)	none present	0 -6.7
8) Heart diseases • aortic or mitral murmur • cardiac failure • cardiomyopathy • atrial fibrillation • cardiomegaly • myocardial infarction within six months	none present	0 -4.3
10) Constant		-12.6
Total		

GREEK STROKE SCORE: [30]

Greek stroke score was devised by a team from Athens. Efstathiou SP and Co-workers proposed a model i.e., Greek score which helps the clinician to diagnose ischemic and haemorrhagic stroke easily. The parameters used in calculating Greek score are easily available to physician soon after admission. A study from Ethiopia showed that the sensitivity, specificity, positive predictive value and negative predictive value is 99%, 99%, 97% and 97% respectively. The above results are much better when compared to Allen and Siriraj scores. Aamod Soman and co-workers from Grant Medical college Hospital, Mumbai reported that the sensitivity, specificity, positive predictive value and negative predictive value is 41%, 95%, 71% and 81% respectively. However the Allen's score can be computed only after 24 hours, so they concluded that Greek score is better than Allen's score. When physician wants to find out the diagnosis at the time of admission, use of Greek score is advisable.

Advantages

1. The clinical parameters used in calculating Greek score are easily memorized and it can be applied at the bedside.
2. The variables used in Greek score are available to the treating physician within first 3 hours of admission.
3. Calculator is not needed.
4. It has high specificity for identifying haemorrhagic stroke.

Variables	Score
1) Neurological deterioration within 3 hours of admission	**6**
2) Vomiting	**4**
3) WBC >12000	**4**
4) Decreased level of consciousness	**3**

The inference of stroke score is based on the below range;

Less than or equal to 3 denotes infarct

4 -12 denotes equivocal

>12 denotes haemorrhage

MATERIALS AND METHODS

SOURCE OF STUDY:

Data consists of primary data collected by the principal investigator directly from the patients admitted with clinical diagnosis of stroke in Coimbatore medical college hospital, Coimbatore.

DESIGN OF STUDY: Cross sectional study

PERIOD OF STUDY: One year; July 2017 – June 2018.

METHODOLOGY:

This is a cross sectional study of patients admitted with clinical diagnosis of stroke in Coimbatore Medical College Hospital, Coimbatore from July 2017 to June 2018.

Inclusion criteria

1) All patients > 20 years presenting with acute stroke i.e rapidly developing symptoms and signs due to focal or global loss of cerebral function lasting for more than 24 hours with no apparent cause other than vascular origin.

Exclusion criteria:

1) Stroke onset more than one week.
2) History of head injury in the past 6 months.
3) Patients on anticoagulants.

4) Patient dying or leaving the hospital in less than 24 hours after admission.

5) CT scan could not be done due to any reason.

6) Patients with bilateral motor weakness.

The data obtained were analyzed using SPSS version 21.0 software. Results were expressed in frequencies and percentages.

Informed consent was obtained from all patients before enrolling in the study.

The study was clearly explained to the patients and relatives in their own language.

OBSERVATION OF THE STUDY AND RESULTS

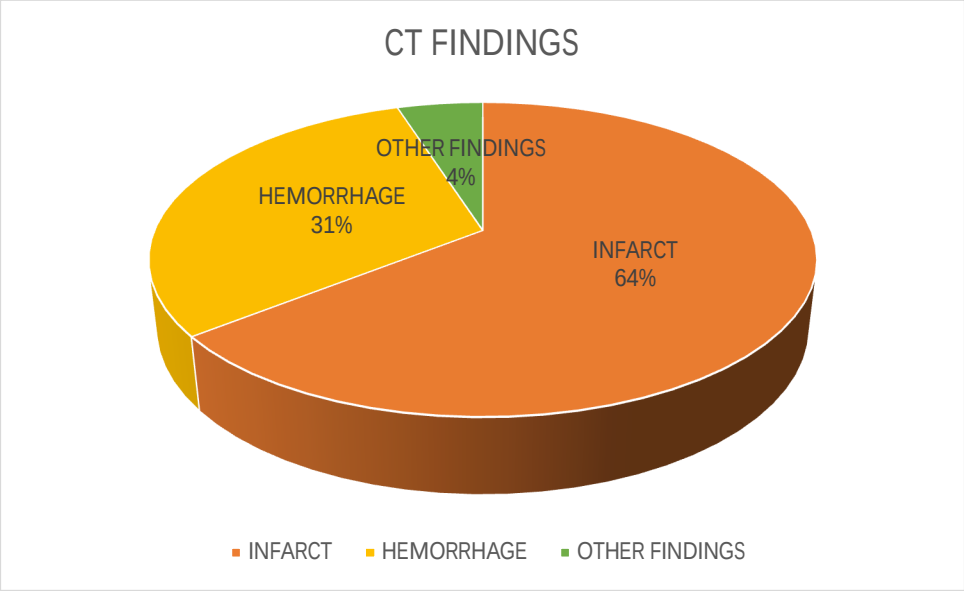
The sample size of our study is 100 patients. All the cases who were clinically confirmed as cerebrovascular accident, has taken CT brain within 24 hours.

Out of 100 cases, 65 patients were proven to be ischemic stroke by CT scan, 31 patients had haemorrhagic stroke.4 out of 100 patients had cerebral venous thrombosis, glioma and tuberculoma. Those four patients were excluded from the study.

Table 1- showing distribution of cases according to CT brain

CT FINDINGS	NO OF PATIENTS	PERCENTAGE
INFARCT	65	65%
HEMORRHAGE	31	31%
OTHER FINDINGS	4	4%

Figure 1-Distribution of cases according to CT brain



AGE DISTRIBUTION:

The age group of the patients in our study group included from 25 to 90 years.

The incidence of stroke was more common in age group of 51-60 years (36%).

The next common age group affected are 41-50years (28%).

Table 2- Age distribution of Stroke cases

AGE IN YEARS	NO OF PATIENTS	PERCENTAGE
< 40	13	13%
41-50	28	28%
51-60	36	36%
61-70	16	16%
>70	7	7%

Figure 2- Age distribution of Stroke cases

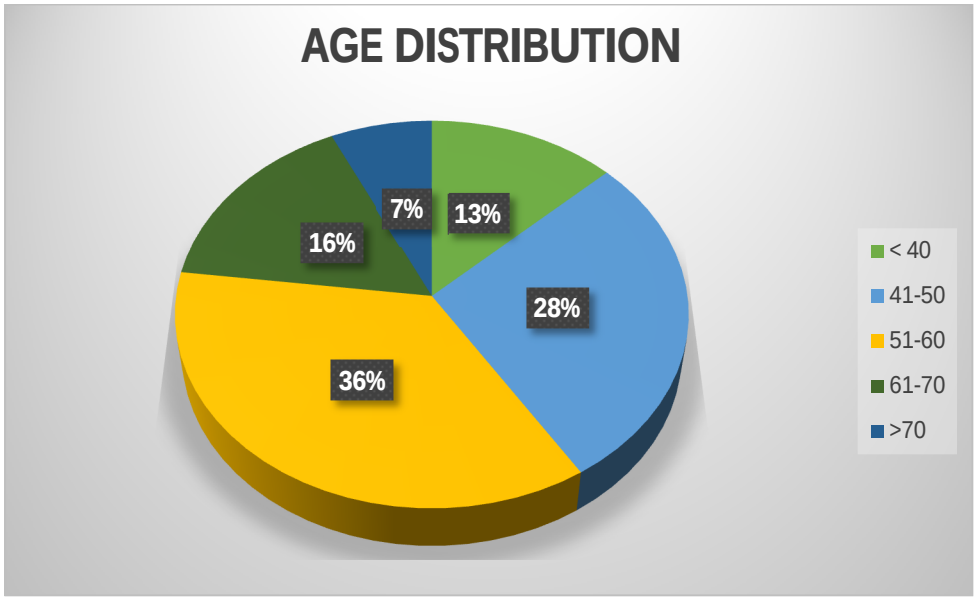
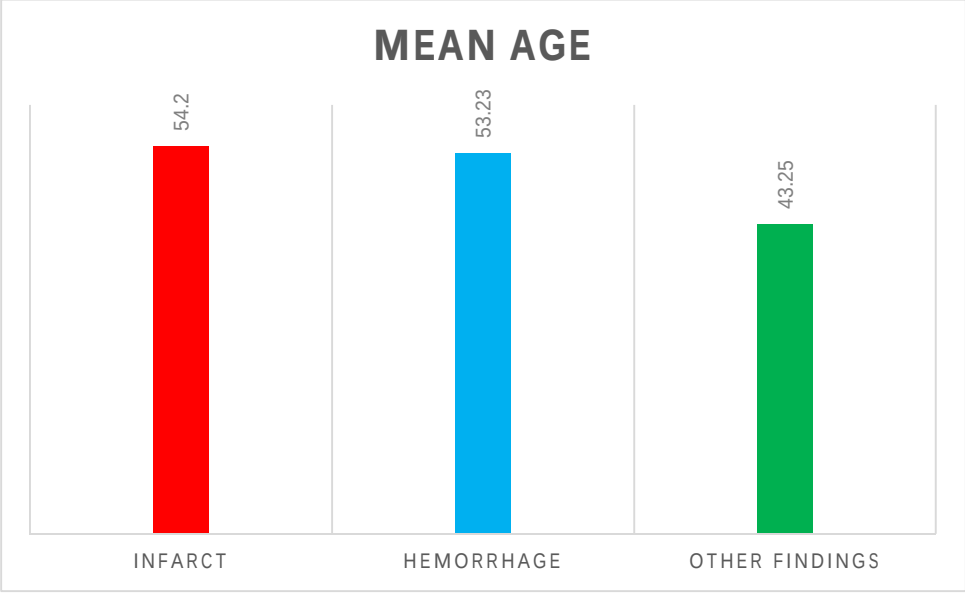


Table 3 showing Independent samples T-test to compare mean age

CT FINDINGS	AGE IN YEARS	
	MEAN	SD
INFARCT	54.2	9.66
HEMORRHAGE	53.23	10.5
OTHER FINDINGS	43.25	9.14
ANOVA		
P VALUE - 0.111		
NON SIGNIFICANT		

Figure 3 Showing Mean Age years



Ischemic stroke and Haemorrhagic stroke are equal incidence among the age group of 40- 60 years (64%).

SEX DISTRIBUTION:

In the group of 96 patients, 19 were male in ischemic stroke group and 46 were females. In haemorrhagic stroke group, 15 were male and 16 were female.

The incidence of ischemic stroke in females outnumbered the male population in this study. In haemorrhagic stroke group, the male and female patients were equally affected.

Table 4 – Showing gender distribution

SEX	NO OF PATIENTS	PERCENTAGE
MALE	35	35%
FEMALE	65	65%

Figure 4 – Showing gender distribution

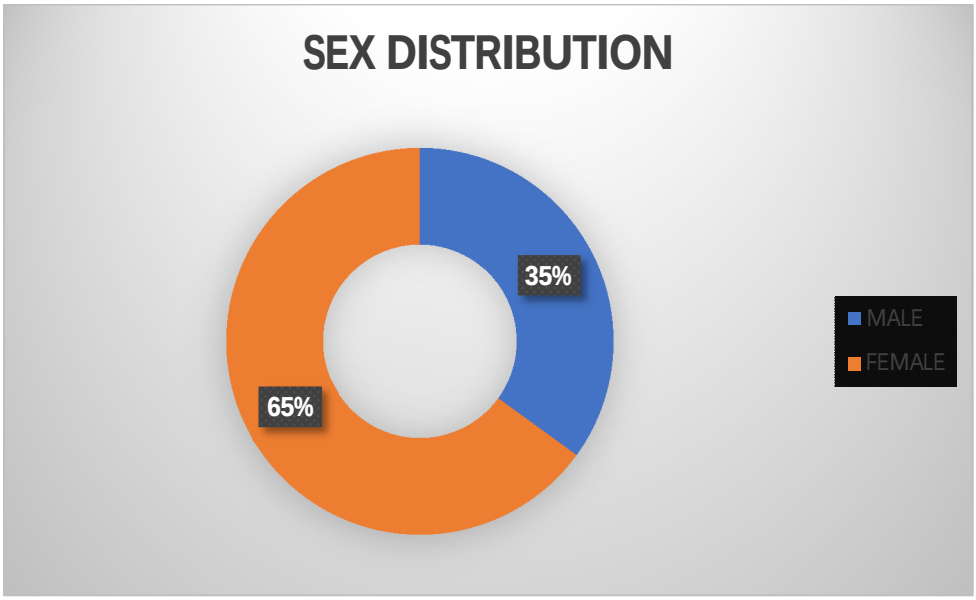


Table 5 – Showing gender distribution among two groups

CT FINDINGS	SEX	
	MALE	FEMALE
INFARCT	19	46
HEMORRHAGE	15	16
OTHER FINDINGS	1	3
KRUSKAL WALLIS TEST		
P VALUE - 0.168		
NON SIGNIFICANT		

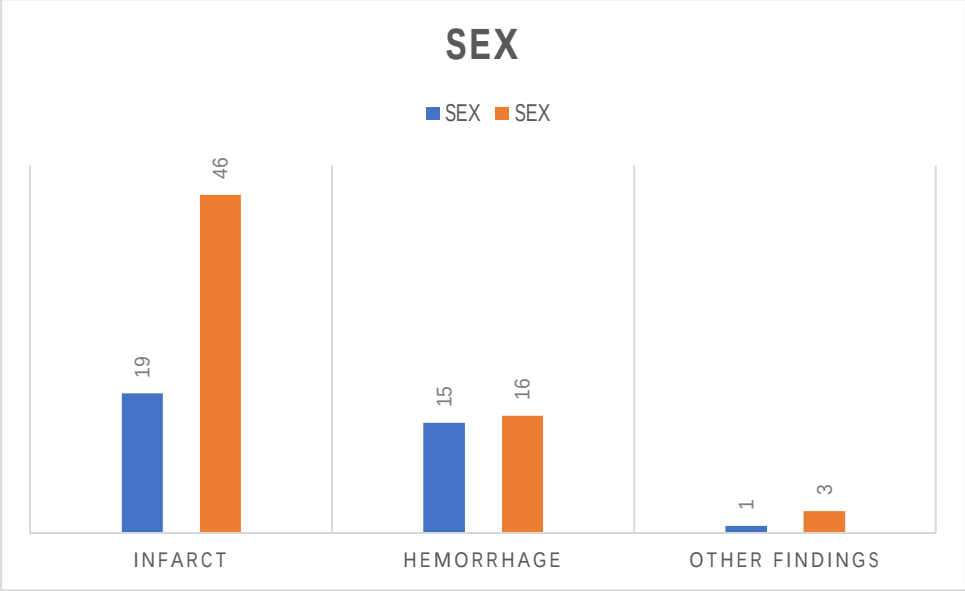


Figure 5 - Showing gender distribution among two groups

VOMITING:

Vomiting is present in 19 patients out of 100 patients in the study

Table 6 – showing percentage of patients with vomiting

VOMITING	NO OF PATIENTS	PERCENTAGE
PRESENT	19	19%
ABSENT	81	81%

Figure 6 – showing percentage of patients with vomiting

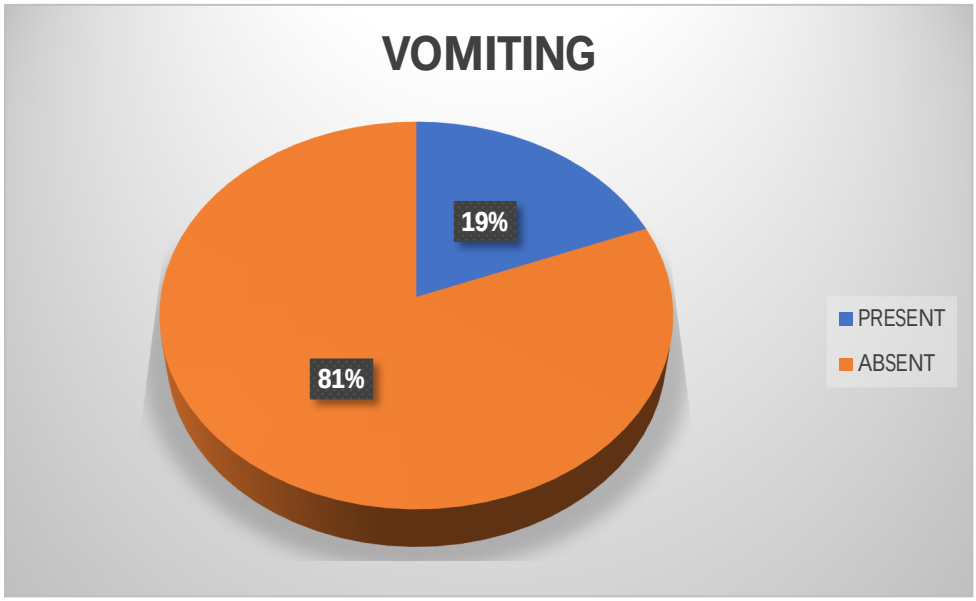
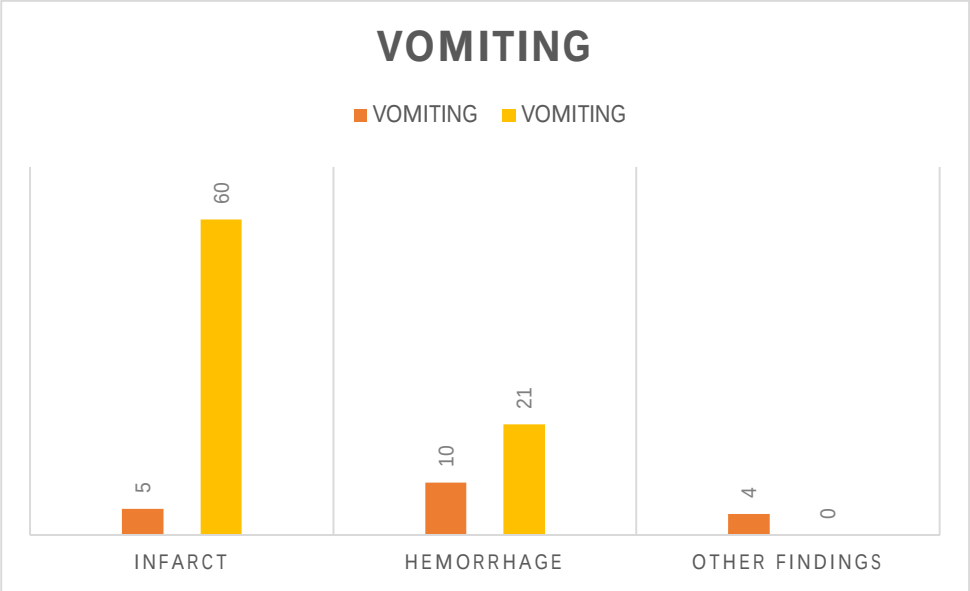


Table 7 – showing distribution of vomiting

CT FINDINGS	VOMITING	
	PRESENT	ABSENT
INFARCT	5	60
HEMORRHAGE	10	21
OTHER FINDINGS	4	0
KRUSKAL WALLIS TEST		
P VALUE - 0.001		
SIGNIFICANT		

At the time of onset of stroke, majority of the patients in the haemorrhagic stroke group had vomiting. Out of 65 patients in ischemic stroke group, 5 patients had vomiting and 10 patients had vomiting in haemorrhagic stroke group.

Figure 7– showing distribution of vomiting



DISTRIBUTION OF HEADACHE:

Table 8 shows percentage of patients who had headache

HEADACHE	NO OF PATIENTS	PERCENTAGE
PRESENT	26	26%
ABSENT	74	74%

Figure 8 shows percentage of patients who had headache

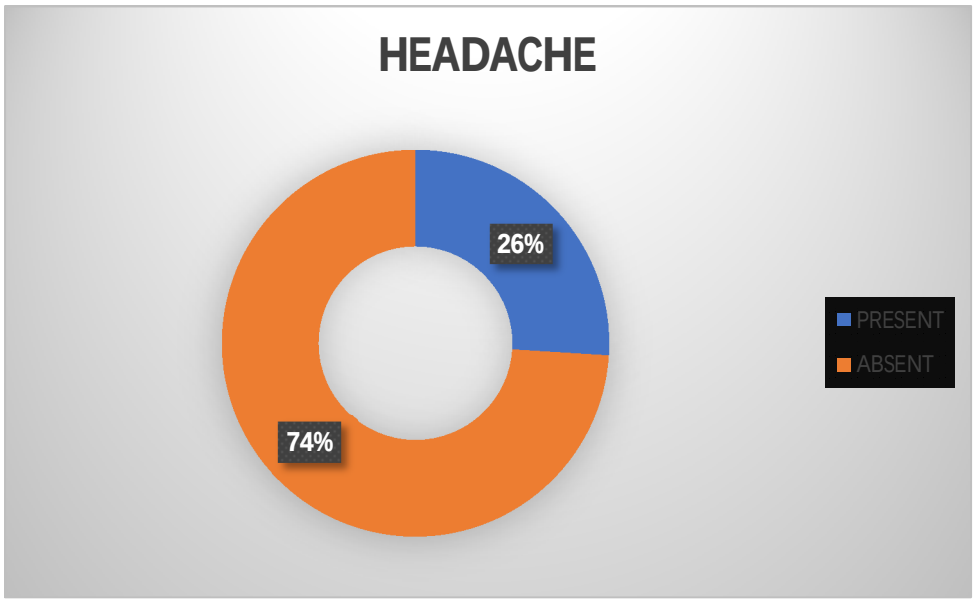
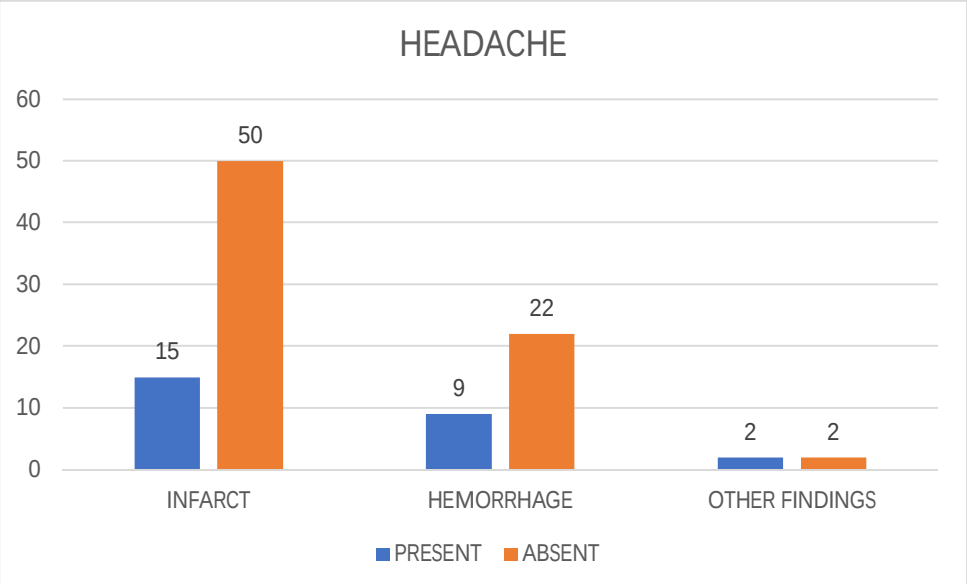


Table 9 shows percentage of patients who had headache in each group

CT FINDINGS	HEADACHE	
	PRESENT	ABSENT
INFARCT	15	50
HEMORRHAGE	9	22
OTHER FINDINGS	2	2
KRUSKAL WALLIS TEST		
P VALUE - 0.442		
NON SIGNIFICANT		

At the time of onset of stroke, majority of the patients had headache. Out of 65 patients in ischemic stroke group, 15 patients had headache and 9 patients had headache in haemorrhagic stroke group.

Figure 9 - shows percentage of patients who had headache in each group



DISTRIBUTION OF HYPERTENSION:

Hypertension is a major modifiable risk factor for ischemic stroke and haemorrhagic stroke. In our study, correlation of ischemic stroke to hypertension is higher. 53 patients were hypertensive in ischemic stroke patients, where 18 patients had hypertension in haemorrhagic stroke group. The p value is 0.006 which is statistically significant.

Table 10 – showing percentage of patients with hypertension

HYPERTENSION	NO OF PATIENTS	PERCENTAGE
PRESENT	72	35%
ABSENT	28	65%

Figure 10 – showing percentage of patients with hypertension

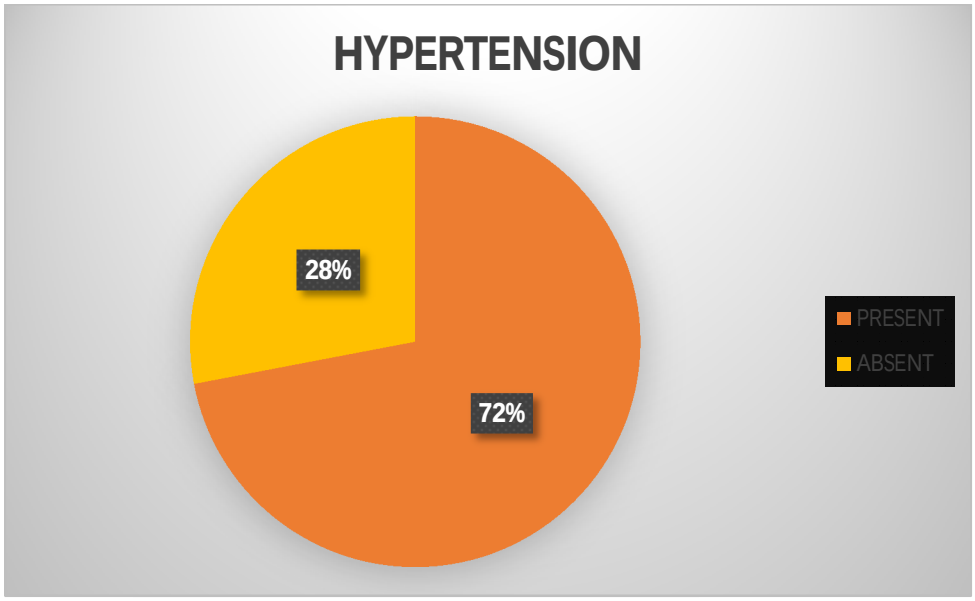
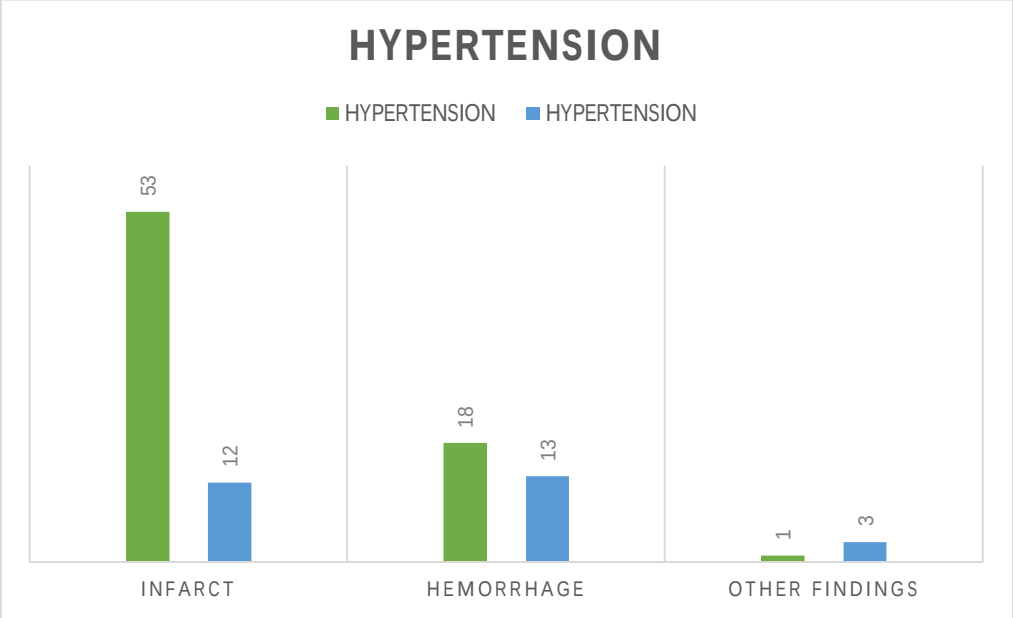


Table 11 – showing distribution of hypertension in two groups

CT FINDINGS	HYPERTENSION	
	PRESENT	ABSENT
INFARCT	53	12
HEMORRHAGE	18	13
OTHER FINDINGS	1	3
KRUSKAL WALLIS TEST		
P VALUE - 0.006		
SIGNIFICANT		

Figure 11– showing distribution of hypertension in two groups

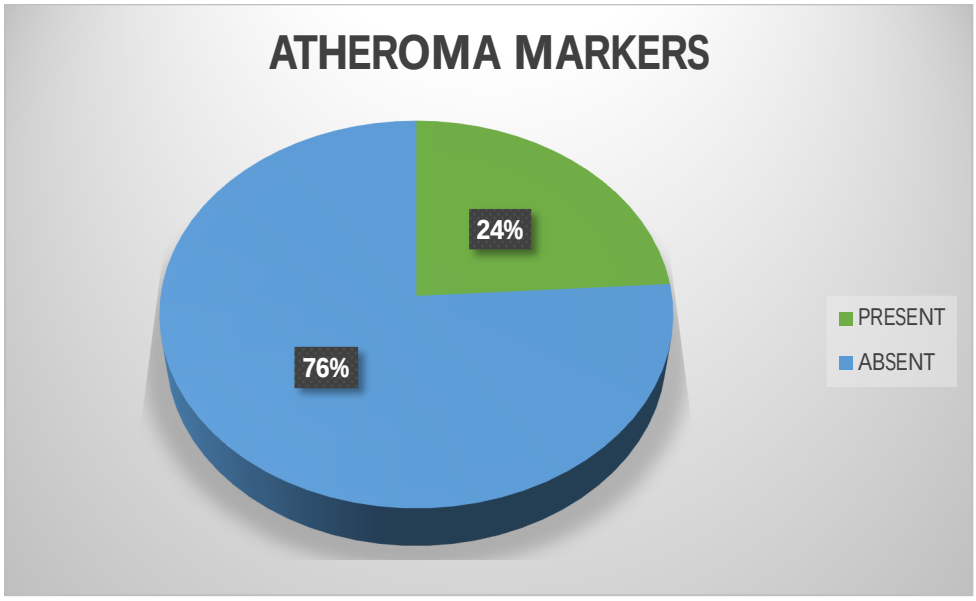


DISTRIBUTION OF ATHEROMA MARKERS

Table 12 – Showing percentage of patients with atheroma markers

ATHEROMA MARKERS	NO OF PATIENTS	PERCENTAGE
PRESENT	24	24%
ABSENT	76	76%

Figure 12 – Showing percentage of patients with atheroma markers

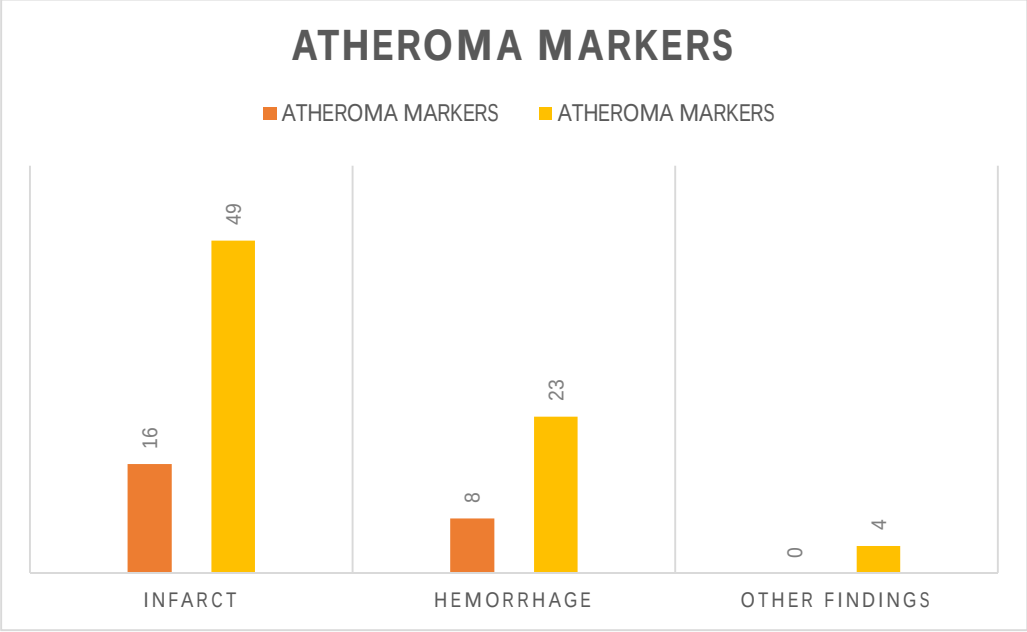


Atheroma markers present in 16 patients in ischemic stroke group and 8 patients in haemorrhagic stroke group. Diabetes mellitus was the common atheroma marker in both ischemic and haemorrhagic stroke population, followed by angina and intermittent claudication.

Table 13 – Showing percentage of atheroma markers in two groups

CT FINDINGS	ATHEROMA MARKERS	
	PRESENT	ABSENT
INFARCT	16	49
HEMORRHAGE	8	23
OTHER FINDINGS	0	4
KRUSKAL WALLIS TEST		
P VALUE - 0.514		
NON SIGNIFICANT		

Figure 13 – Showing percentage of atheroma markers in two groups

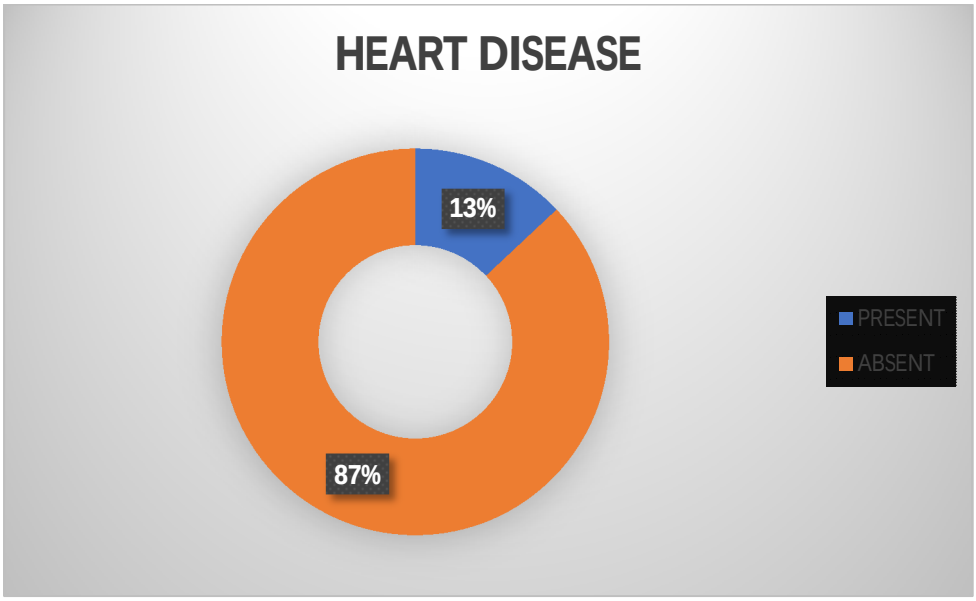


DISTRIBUTION OF HEART DISEASE:

Table 14 – shows percentage of patients with heart disease

HEART DISEASE	NO OF PATIENTS	PERCENTAGE
PRESENT	13	13%
ABSENT	87	87%

Figure 14– shows percentage of patients with heart disease

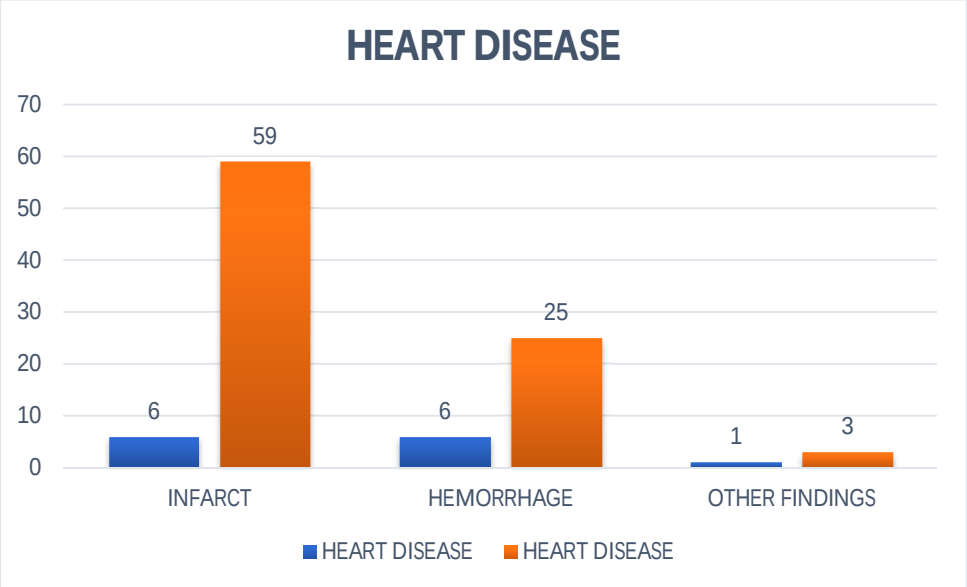


Heart diseases were present in 12 out of 96 patients in the study population. 3 patients had Rheumatic heart disease, 8 patients had coronary artery disease and one patient had atrial fibrillation.

Table 15 – shows percentage of heart disease in two groups

CT FINDINGS	HEART DISEASE	
	PRESENT	ABSENT
INFARCT	6	59
HEMORRHAGE	6	25
OTHER FINDINGS	1	3
KRUSKAL WALLIS TEST		
P VALUE - 0.296		
NON SIGNIFICANT		

Figure 15 – shows percentage of heart disease in two groups

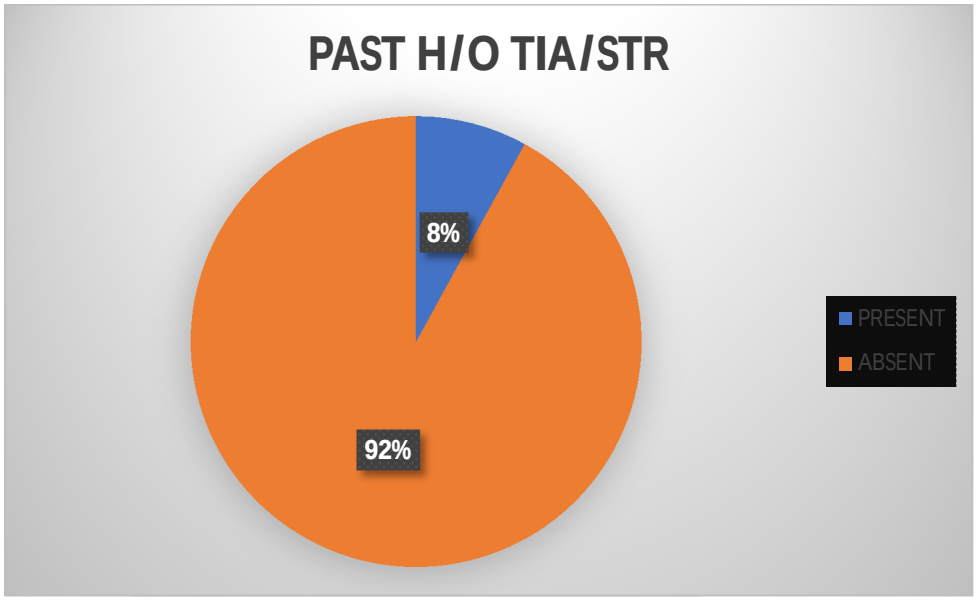


DISTRIBUTION OF TIA/STROKE:

Table 16 - Showing percentage of patients with past History of TIA/Stroke

PAST H/O OF TIA/STR	NO OF PATIENTS	PERCENTAGE
PRESENT	8	8%
ABSENT	92	92%

Figure 16 - Showing percentage of patients with past History of TIA/Stroke

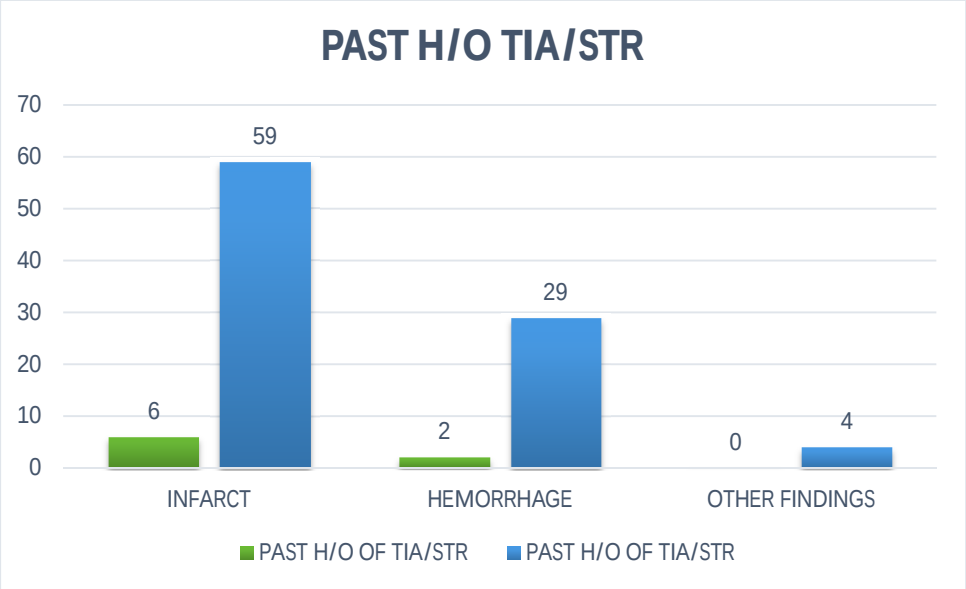


Past history of stroke or transient ischemic attack were present in 8 out of 96 patients in the study population.

Table 17 - Showing percentage of past History of TIA/Stroke in two groups

CT FINDINGS	PAST H/O OF TIA/STR	
	PRESENT	ABSENT
INFARCT	6	59
HEMORRHAGE	2	29
OTHER FINDINGS	0	4
KRUSKAL WALLIS TEST		
P VALUE - 0.747		
NON SIGNIFICANT		

Figure 17 - Showing percentage of past History of TIA/Stroke in two groups



DISTRIBUTION OF SMOKING:

Table 18 - Showing percentage of patients with smoking

SMOKING	NO OF PATIENTS	PERCENTAGE
PRESENT	25	25%
ABSENT	75	75%

Figure 18 - Showing percentage of patients with smoking

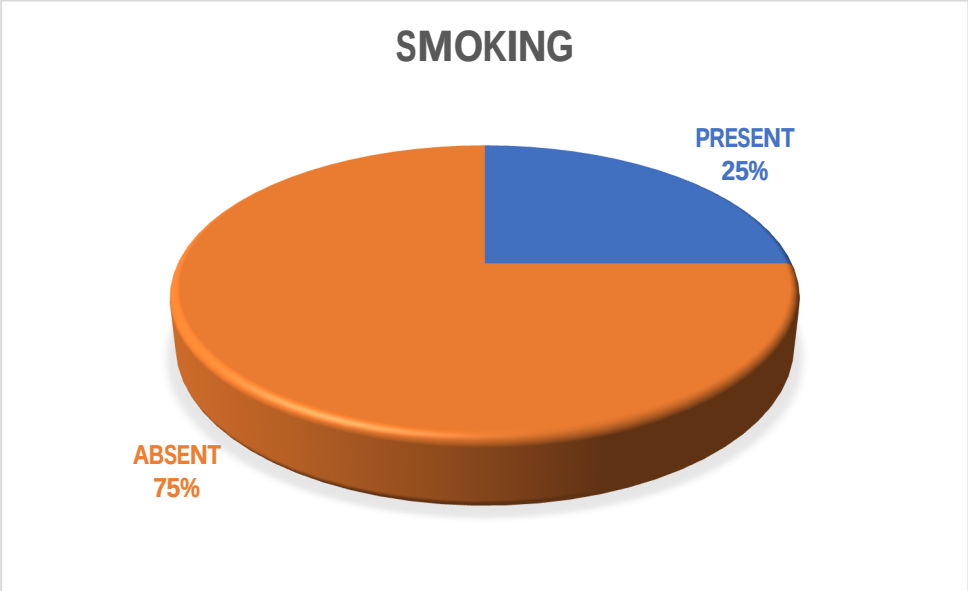
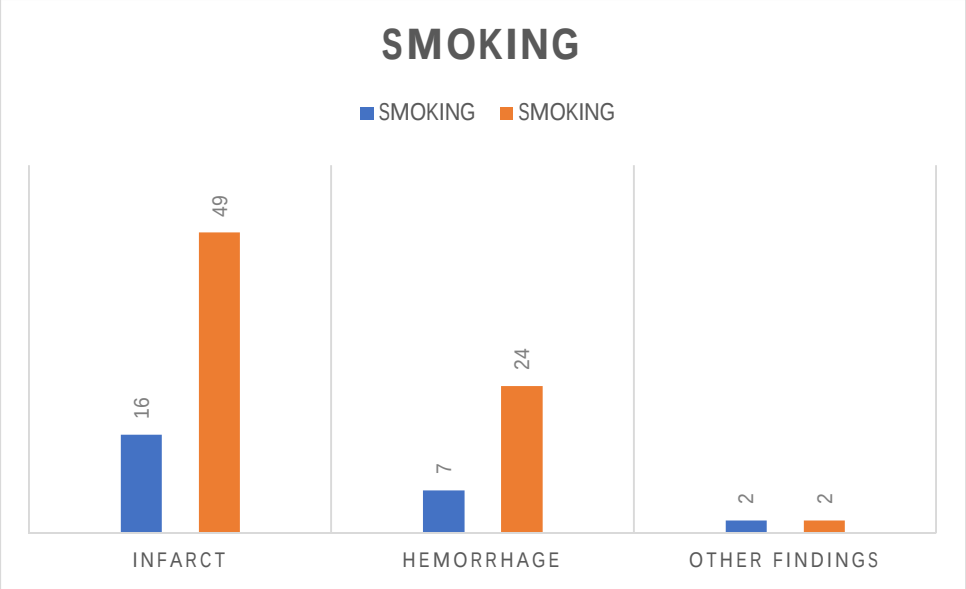


Table 19 - Showing percentage of patients with smoking in both groups

CT FINDING	SMOKING	
	PRESENT	ABSENT
INFARCT	16	49
HEMORRHAGE	7	24
OTHER FINDINGS	2	2
KRUSKAL WALLIS TEST		
P VALUE - 0.488		
NON SIGNIFICANT		

Smoking is a modifiable risk factor for stroke. Smoking is present in 25% of 96 patients in the study. 16 patients were smokers in ischemic stroke group and 7 patients were smokers in haemorrhagic stroke group.

Figure 19 - Showing percentage of patients with smoking in both groups



DISTRIBUTION OF ALCOHOL INTAKE:

Table 20 – showing percentage of patients with history of alcohol intake

ALCOHOL	NO OF PATIENTS	PERCENTAGE
PRESENT	54	54%
ABSENT	46	46%

Figure 20 – showing percentage of patients with history of alcohol intake

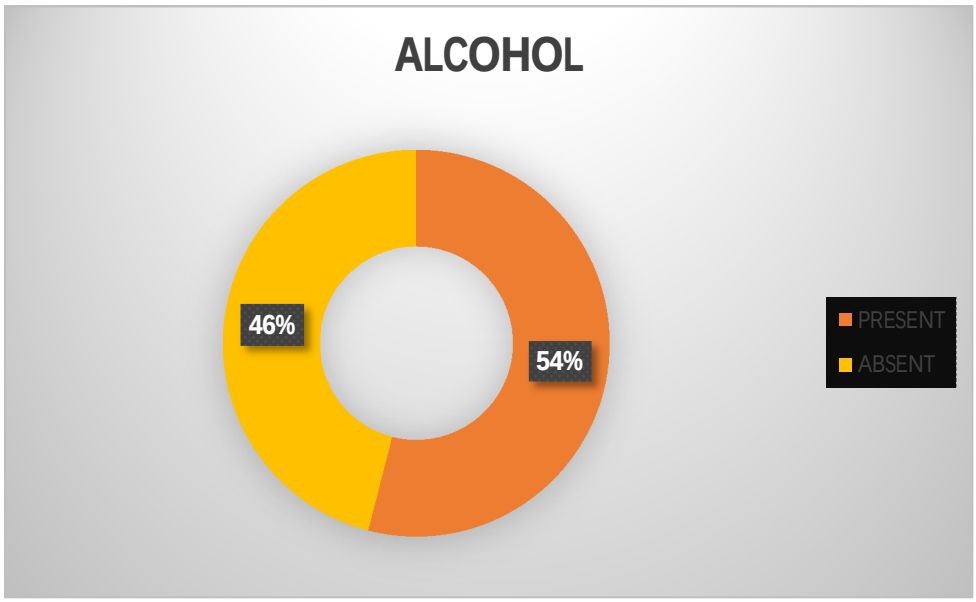
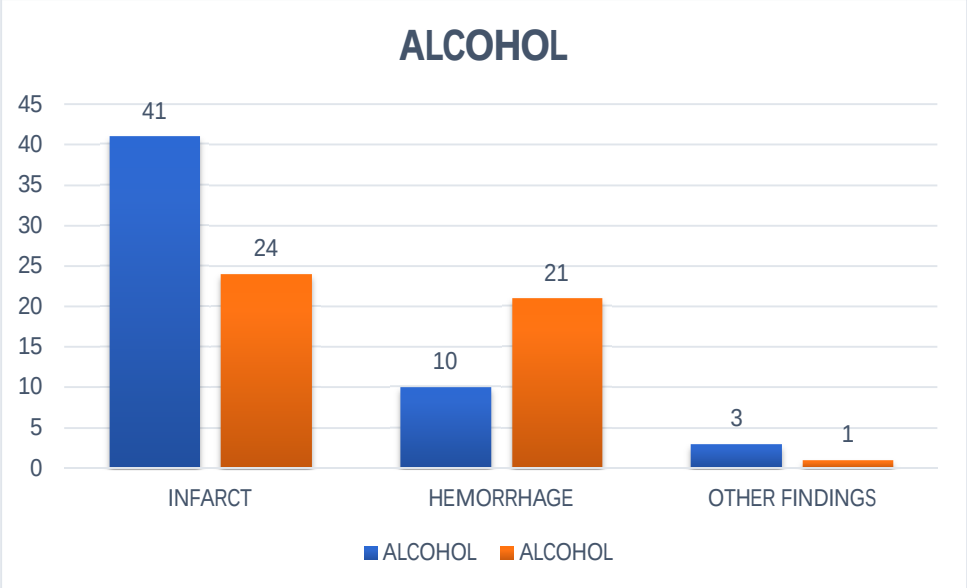


Table 21 – showing distribution of alcohol intake

CT FINDING	ALCOHOL	
	PRESENT	ABSENT
INFARCT	41	24
HEMORRHAGE	10	21
OTHER FINDINGS	3	1
KRUSKAL WALLIS TEST		
P VALUE - 0.012		
SIGNIFICANT		

History of alcohol intake was present in 51 out of 96 patients in this study. The incidence of Ischemic stroke was common in alcoholics when compared to non-alcoholics. The p value is 0.012, which is statistically significant.

Figure 21 – showing distribution of alcohol intake



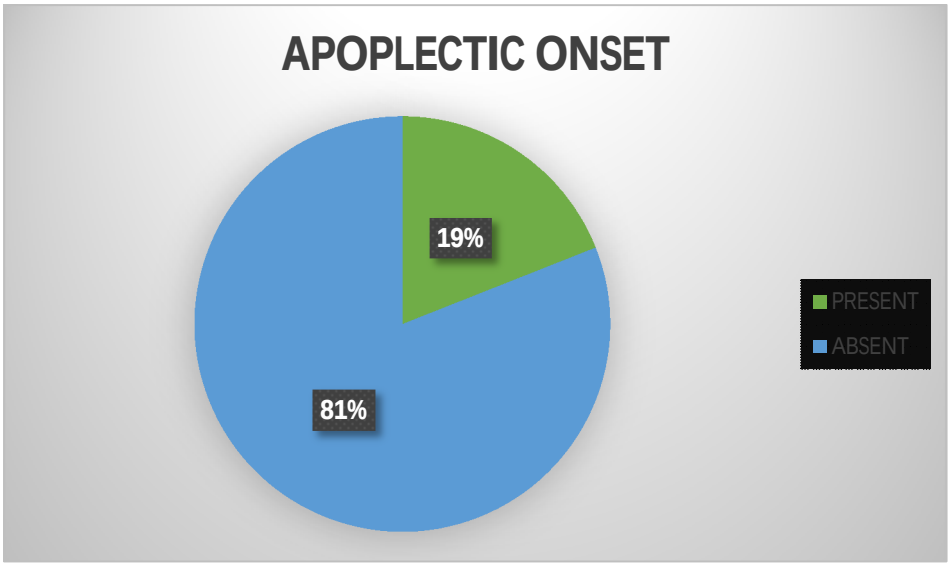
DISTRIBUTION OF APOPLECTIC ONSET:

Apoplectic onset means when any two of four findings was present, the findings are headache within 2 hours, headache, vomiting and loss of consciousness.

Table 22 – showing percentage of patients with apoplectic onset

APOPLECTIC ONSET	NO OF PATIENTS	PERCENTAGE
PRESENT	19	19%
ABSENT	81	81%

Figure 22 – showing percentage of patients with apoplectic onset

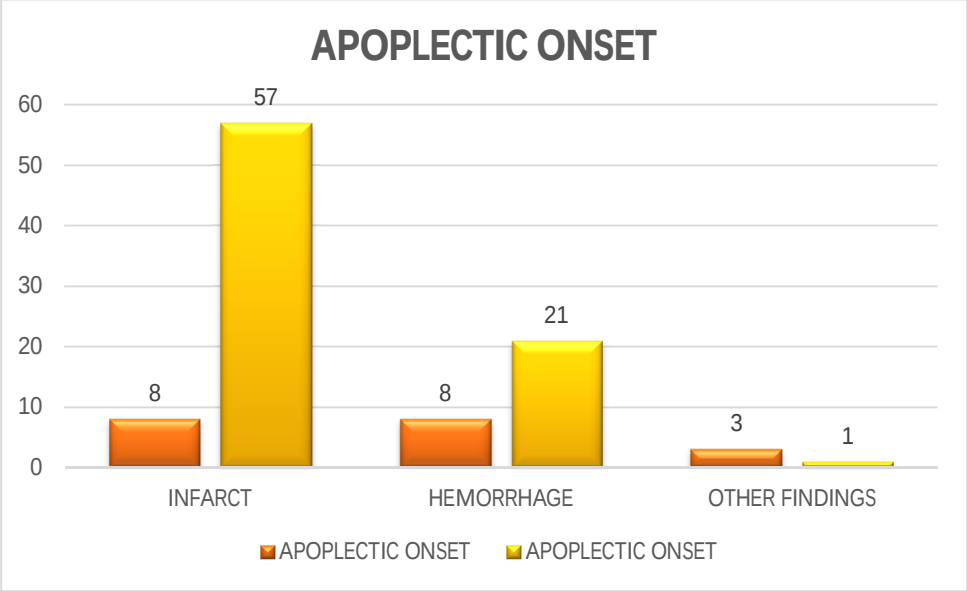


It was present in 8 patients in ischemic stroke group. In patients in haemorrhagic stroke group, 8 out of 31 patients had apoplectic onset. The statistical analysis showed that the p value is 0.004, which is significant.

Table 23 – showing distribution of Apoplectic onset in both groups

CT FINDINGS	APOPLECTIC ONSET	
	PRESENT	ABSENT
INFARCT	8	57
HEMORRHAGE	8	21
OTHER FINDINGS	3	1
KRUSKAL WALLIS TEST		
P VALUE - 0.004		
SIGNIFICANT		

Figure 23 – showing distribution of Apoplectic onset in both groups



DISTRIBUTION OF CONSCIOUS LEVEL AT THE TIME OF ADMISSION:

Table 24 – showing conscious level of patients on admission

CONDITION ON ADMISSION	NO OF PATIENTS	PERCENTAGE
ALERT	57	57%
DROWSY	24	24%
UNCONSCIOUS	19	19%

Figure 24 – showing conscious level of patients at admission

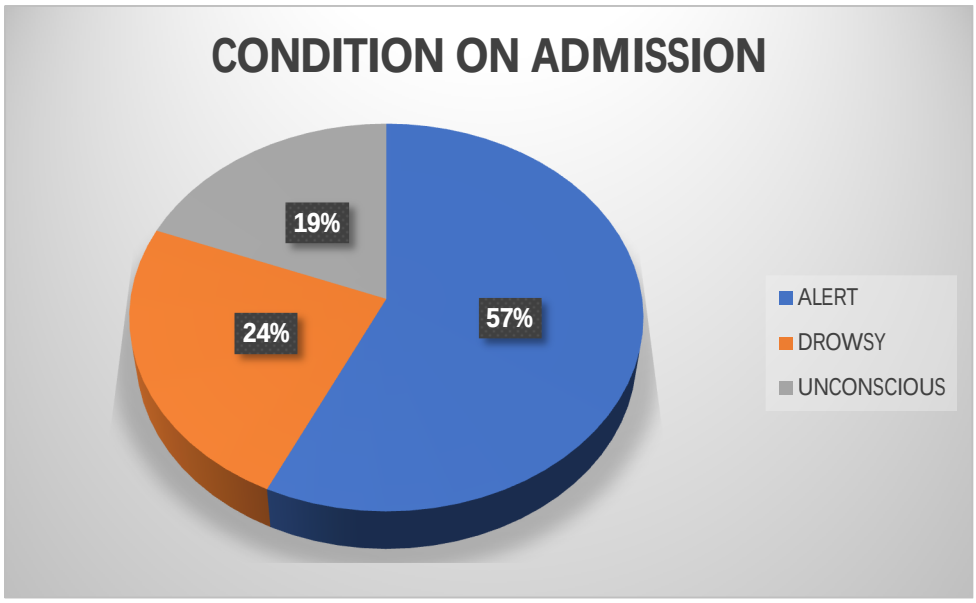
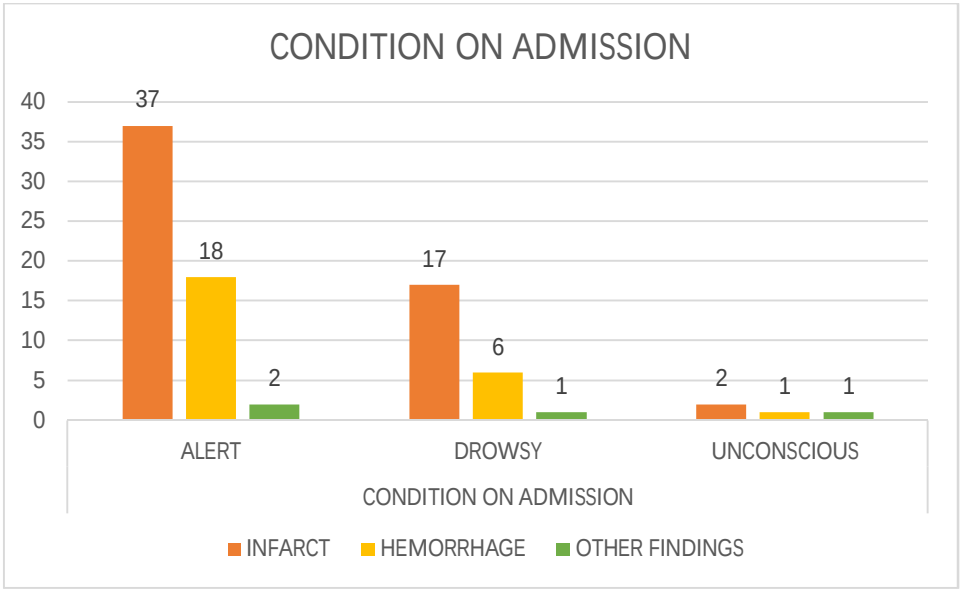


Table 25 – distribution of conscious level on admission

CT FINDINGS	CONDITION ON ADMISSION		
	ALERT	DROWSY	UNCONSCIOUS
INFARCT	37	17	2
HEMORRHAGE	18	6	1
OTHER FINDINGS	2	1	1
KRUSKAL WALLIS TEST			
P VALUE - 0.928			
NON SIGNIFICANT			

In ischemic stroke group patients, 37 patients were alert on admission, 17 patients were drowsy and 2 patients were unconscious at the time of admission. In patients with haemorrhagic stroke, one patient was unconscious, 6 patients were drowsy and rest of the patients were alert on admission.

Figure 25– showing distribution of conscious level on admission

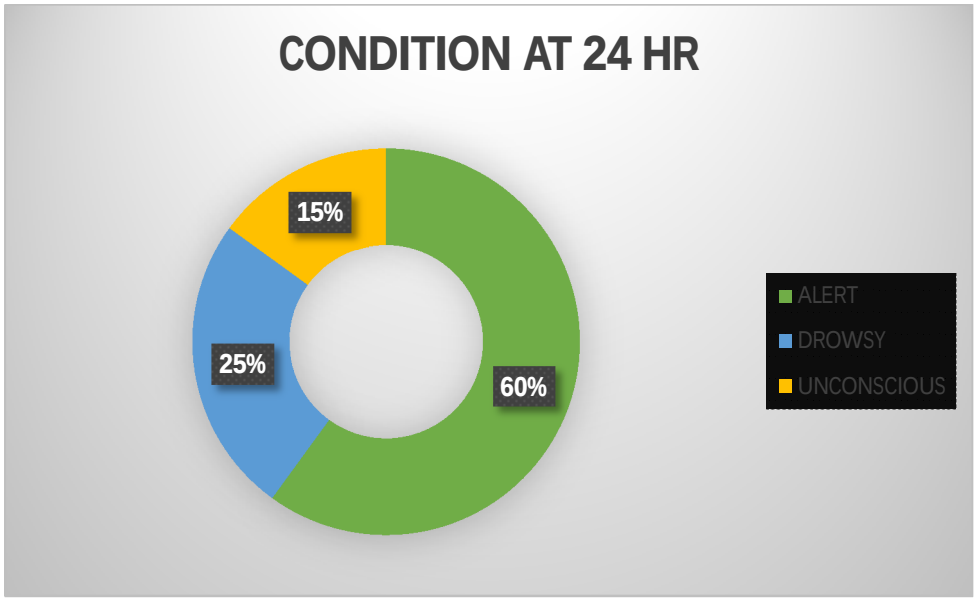


DISTRIBUTION OF CONSCIOUS LEVEL AT 24 HOURS:

Table 26– showing conscious level of patients at 24 hours

CONDITION AT 24 HR	NO OF PATIENTS	PERCENTAGE
ALERT	60	60%
DROWSY	25	25%
UNCONSCIOUS	15	15%

Figure 26 – showing conscious level of patients at 24 hours

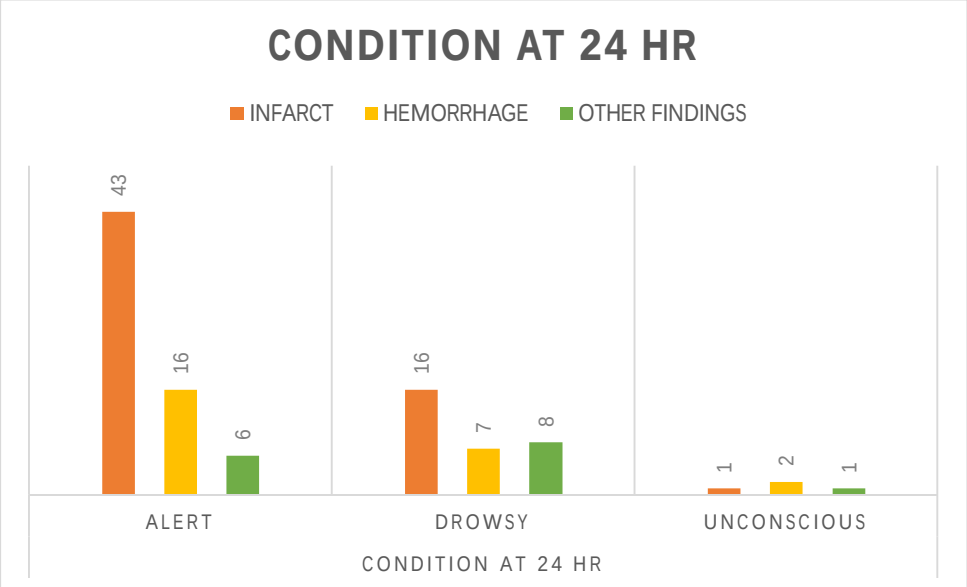


After 24 hours of admission, 43 patients were alert, 16 patients were drowsy and one patient was unconscious in ischemic stroke group. In haemorrhagic stroke group, 16 patients were alert, 7 patients were drowsy and 2 patients are unconscious.

Table 27 – showing distribution of conscious level at 24 hours

CT FINDINGS	CONDITION AT 24 HR		
	ALERT	DROWSY	UNCONSCIOUS
INFARCT	43	16	1
HEMORRHAGE	16	7	2
OTHER FINDINGS	6	8	1
KRUSKAL WALLIS TEST			
P VALUE - 0.147			
NON SIGNIFICANT			

Figure 27 – showing distribution of conscious level at 24 hours

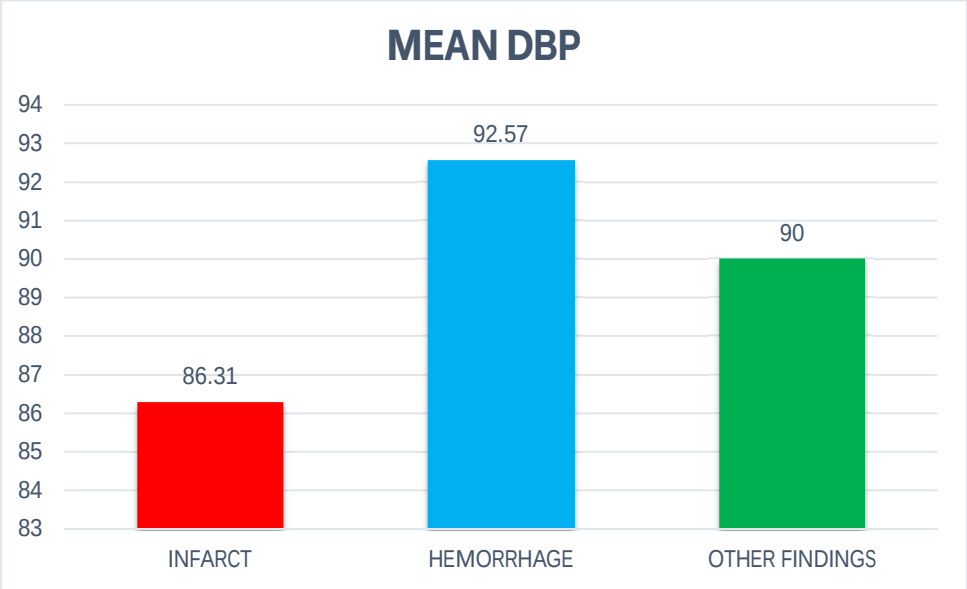


DISTRIBUTION OF DIASTOLIC BLOOD PRESSURE:

Table - 28 shows mean diastolic blood pressure in both groups

CT FINDINGS	DIASTOLIC BP	
	MEAN	SD
INFARCT	86.31	13.05
HEMORRHAGE	92.57	13.65
OTHER FINDINGS	90	0
ANOVA		
P VALUE - 0.328		
NON SIGNIFICANT		

Figure - 28 shows mean diastolic blood pressure in both groups



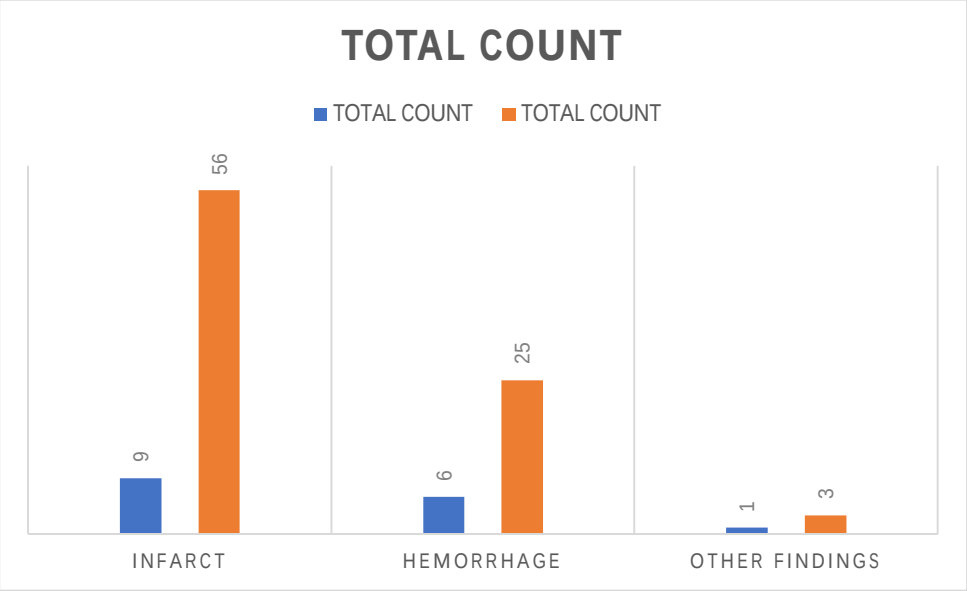
DISTRIBUTION OF TOTAL COUNT:

Table – 29 Shows distribution of WBC count in two Groups

CT FINDINGS	TOTAL COUNT	
	> 12000	**< 12000**
INFARCT	9	56
HEMORRHAGE	6	25
OTHER FINDINGS	1	3
KRUSKAL WALLIS TEST		
P VALUE - 0.696		
NON SIGNIFICANT		

White cell count is increased in 9 patients out of 65 patients in ischemic stroke group. In haemorrhagic stroke group, 6 patients found to have elevated total count and rest of them had total count less than 12000.

Figure - 29 Shows distribution of WBC count in two Groups



DISTRIBUTION OF PLANTAR RESPONSE:

In ischemic stroke group, 15 patients out of 65 had bilateral plantar extensor response and 10 patients out of 31 had bilateral plantar extensor response in haemorrhagic stroke group. The patients who had bilateral plantar extensor response showed the worse prognosis.

Table – 30 showing distribution of plantar response

CT FINDINGS	PLANTAR RESPONSE		
	B/L FLEXOR	U/L EXTENSOR	B/L EXTENSOR
INFARCT	33	17	15
HEMORRHAGE	15	6	10
OTHER FINDINGS	3	1	0
KRUSKAL WALLIS TEST			
P VALUE - 0.618			
NON SIGNIFICANT			

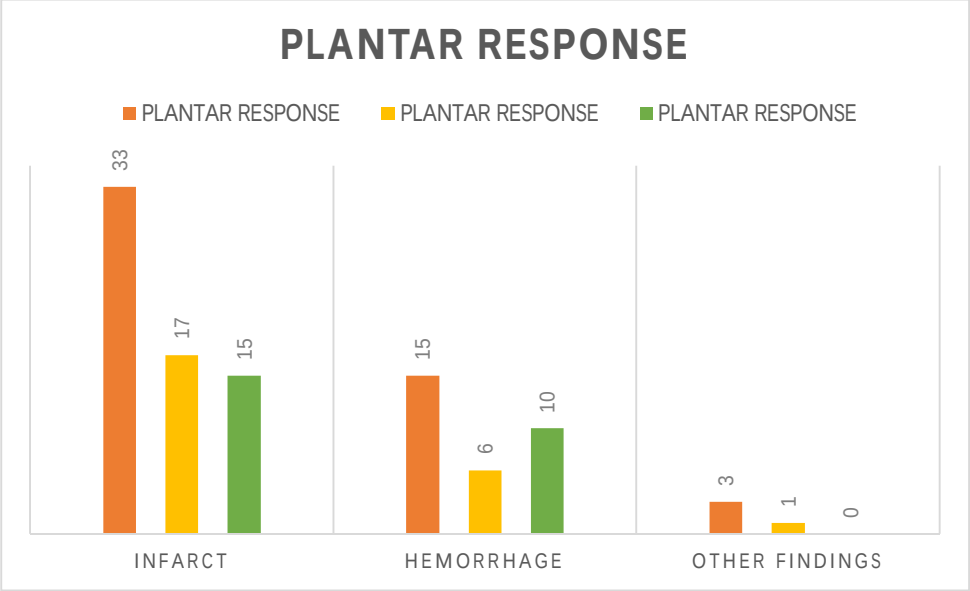


Figure 30 - showing distribution of plantar response

ANALYSIS OF ALLEN'S STROKE SCORE

In our study, 65 patients had infarct which was proven by CT scan brain. Out of 65, Allen stroke score diagnosed 42 patients correctly as having infarct, it also misdiagnosed one patient as having haemorrhage who actually had infarct in CT scan brain. Allen stroke score was equivocal in 22 patients.

Table – 30 showing distribution of plantar response

CT FINDINGS	ALLEN STROKE SCORE		
	≤ 4 (INFARCT)	**5 -24 (EQUIVOCAL)**	**> 24 (HEMORRHAGE)**
INFARCT	42	22	1
HEMORRHAGE	13	6	12

31 patients had haemorrhage which was proven by CT brain. Out of 31, Allen stroke score diagnosed 12 patients correctly as having haemorrhage. However this score wrongly diagnosed 13 patients as having infarct who actually had haemorrhage in CT scan brain.

Allen stroke score diagnosed 56 patients as having infarct, 24 patients as having haemorrhage and equivocal in 15 patients.

Figure 31 – number of patients categorised with Allen stroke score

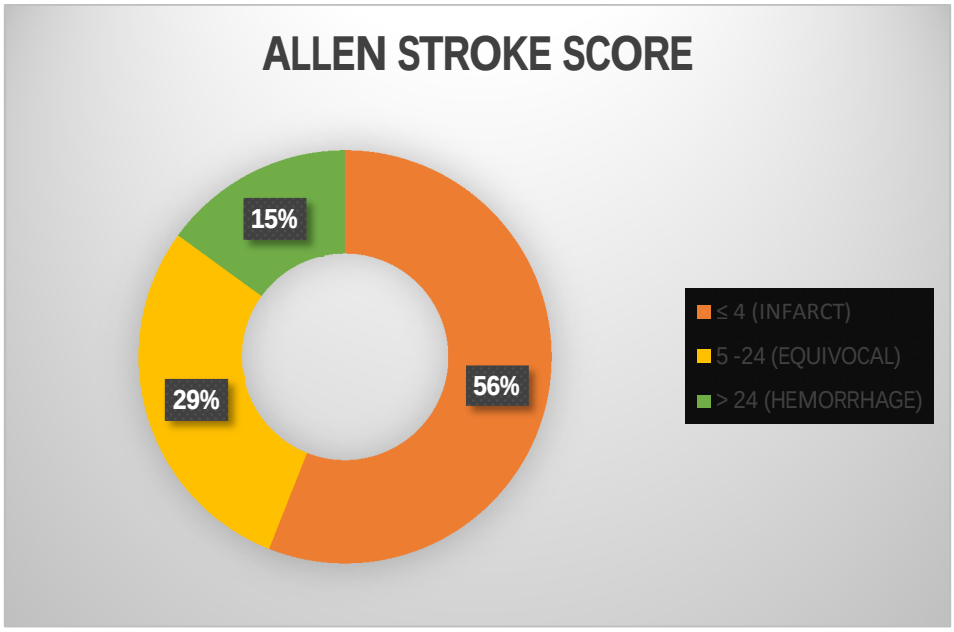
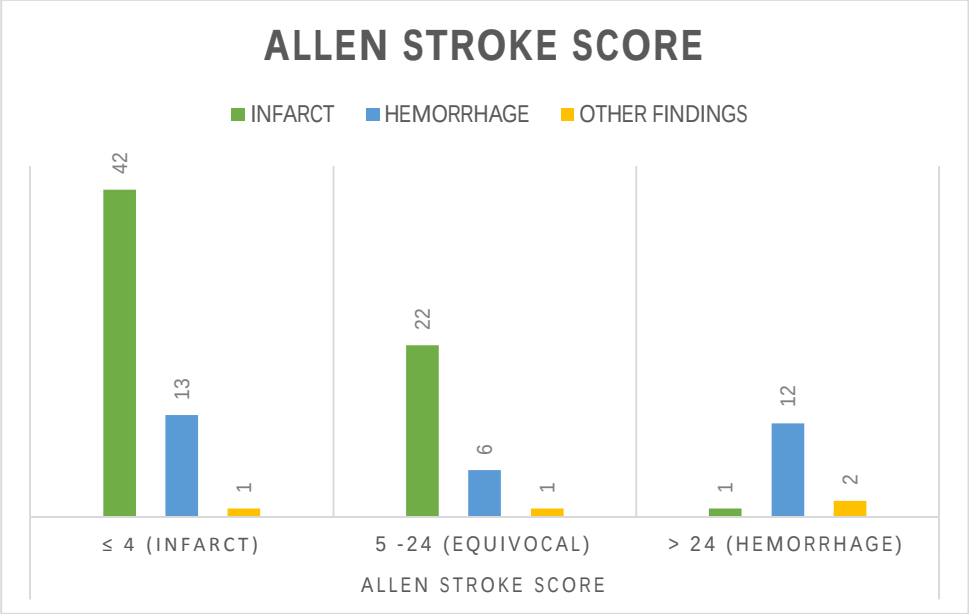


Figure 32- comparison of CT brain with Allen stroke score



Allen stroke score has 64.62 % sensitivity and 60.00 % specificity for infarction. The positive predictive value is 75% and the negative predictive is 47.73 % for infarction. The diagnostic accuracy of Allen's score for diagnosing infarct is 63%.

Table 32 - Evaluation of Allen score with statistical parameters for Infarct

INFARCT	ALLEN STROKE SCORE
SENSITIVITY	64.62%
SPECIFICITY	60.00%
POSITIVE PREDICTIVE VALUE	75.00%
NEGATIVE PREDICTIVE VALUE	47.73%
ACCURACY	63.00%

Figure 33 - Evaluation of Allen score with statistical parameters for Infarct

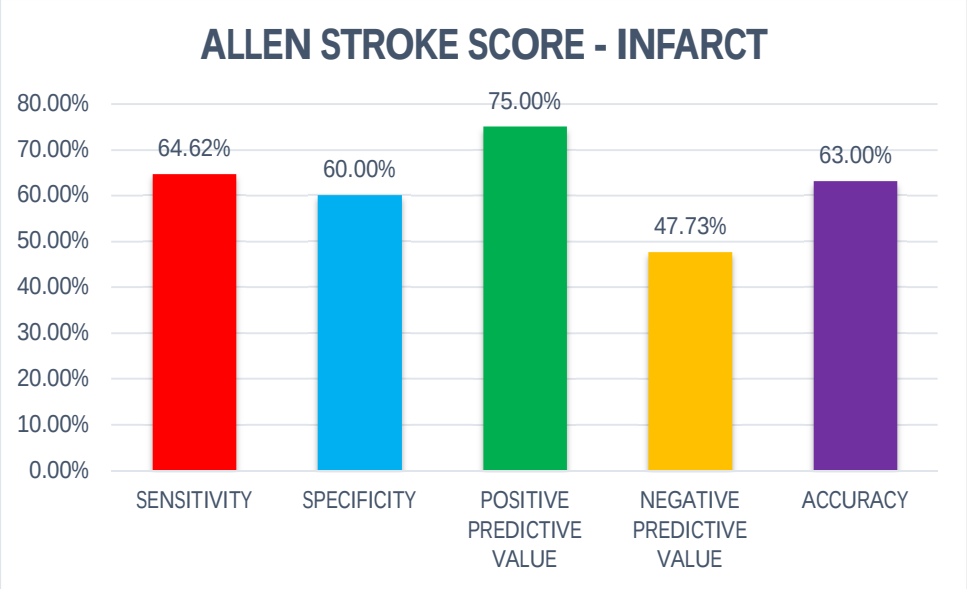
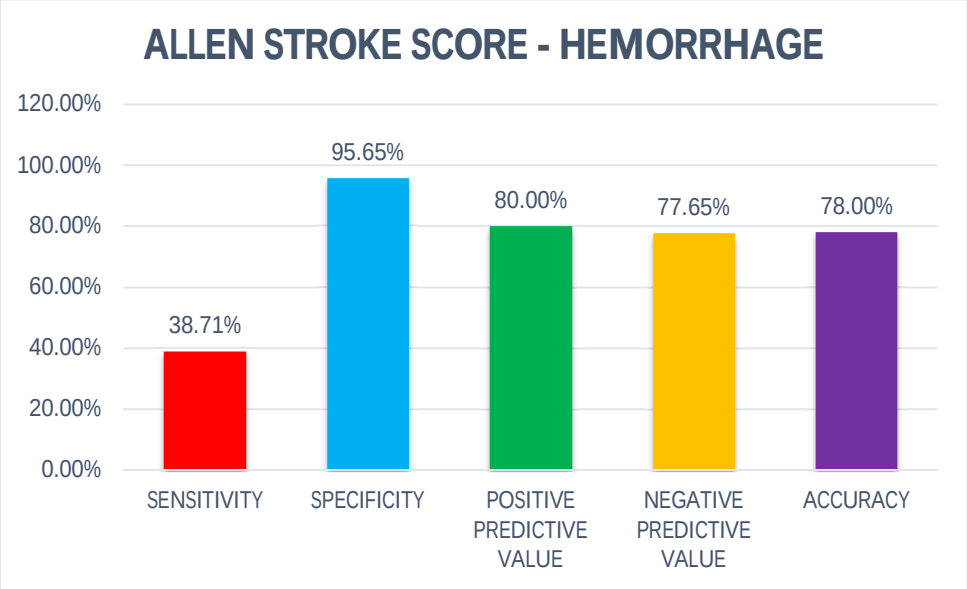


Table 33 - Evaluation of Allen score with statistical parameters for Haemorrhage

HEMORRHAGE	ALLEN STROKE SCORE
SENSITIVITY	38.71%
SPECIFICITY	95.65%
POSITIVE PREDICTIVE VALUE	80.00%
NEGATIVE PREDICTIVE VALUE	77.65%
ACCURACY	78.00%

Figure 34 - Evaluation of Allen score with statistical parameters for Haemorrhage



Allen stroke score has a sensitivity of 38.71 % and 95.65 % for haemorrhage. It also has the positive predictive value of 80 % and negative predictive of 77.65% for haemorrhage. The diagnostic accuracy is 78%.

ANALYSIS OF GREEK STROKE SCORE:

Greek stroke score diagnosed 59 patients as having infarct, 31 patients as having haemorrhage and equivocal in 10 patients.

Figure 35 – number of patients categorised with Greek stroke score

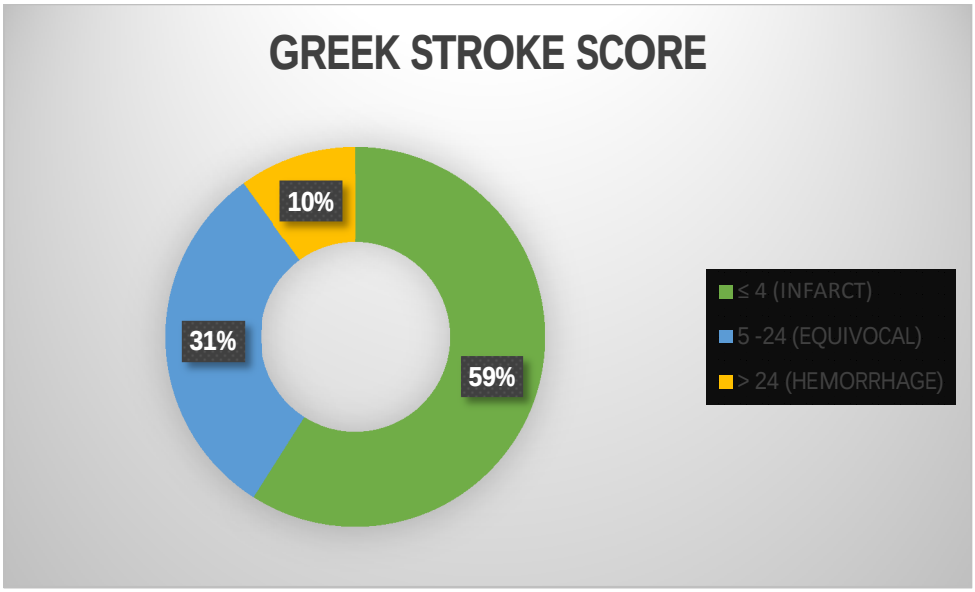


Table 34 - comparison of CT brain with Greek stroke score

CT FINDINGS	GREEK STROKE SCORE		
	≤ 4 (INFARCT)	5 -24 (EQUIVOCAL)	> 24 (HEMORRHAGE)
INFARCT	48	16	1
HEMORRHAGE	10	13	8

Figure 36 - comparison of CT brain with Greek stroke score

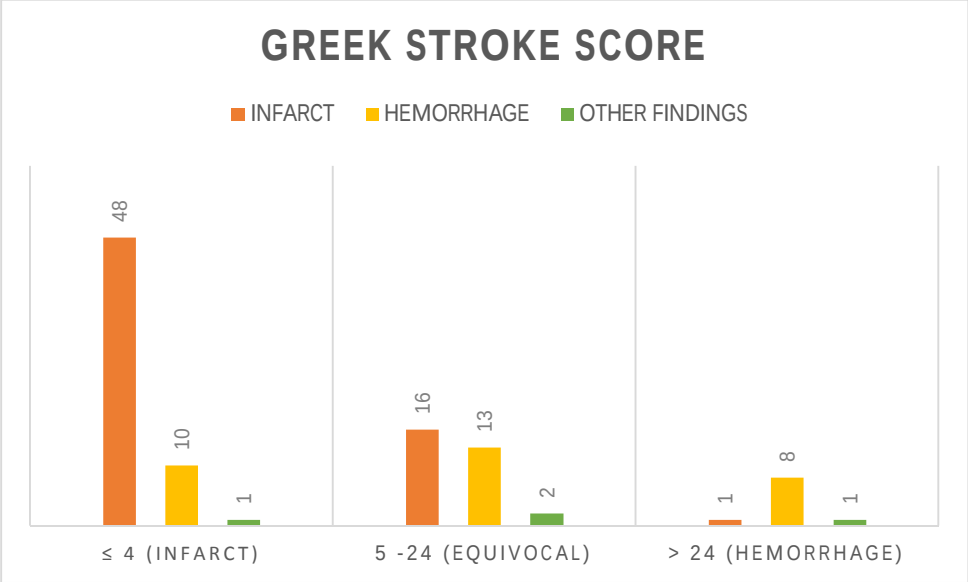


Table 35 - Evaluation of Greek score with statistical parameters for Infarct

INFARCT	GREEK STROKE SCORE
SENSITIVITY	73.85%
SPECIFICITY	68.57%
POSITIVE PREDICTIVE VALUE	81.36%
NEGATIVE PREDICTIVE VALUE	58.54%
ACCURACY	72.00%

The sensitivity of Greek stroke score for infarct is 73.85% and specificity of Greek stroke score for infarct is 68.57%. Its positive predictive value is 81.36% and negative predictive value is 58.54%. The diagnostic accuracy of Greek stroke score for detecting ischemic stroke is 72%.

Figure 37- Evaluation of Greek score with statistical parameters for infarct

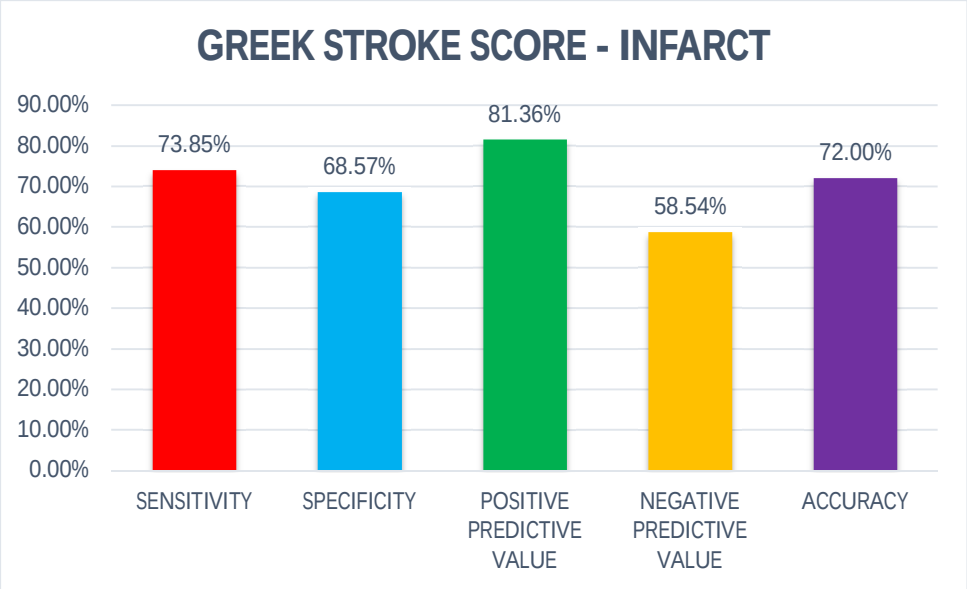
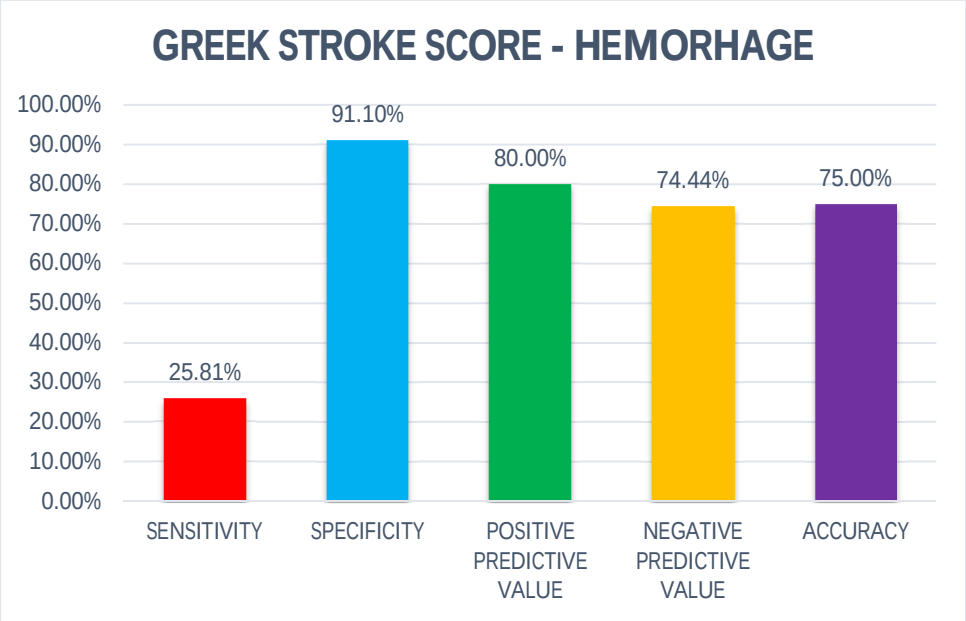


Table-36 Evaluation of Greek score with statistical parameters for Haemorrhage

HEMORRHAGE	GREEK STROKE SCORE
SENSITIVITY	25.81%
SPECIFICITY	91.10%
POSITIVE PREDICTIVE VALUE	80.00%
NEGATIVE PREDICTIVE VALUE	74.44%
ACCURACY	75.00%

The sensitivity of Greek stroke score for Haemorrhage is 25.81% which is lower than other studies. The specificity of Greek stroke score for haemorrhage is 91.10%.Its positive predictive value is 80% and negative predictive value is 74.44%. The diagnostic accuracy of Greek stroke score for detecting Haemorrhage is 75%.

Figure-38 Evaluation of Greek score with statistical parameters for Haemorrhage



DISCUSSION

Management of stroke mainly depends on differentiation of haemorrhagic from ischemic stroke. Clinical stroke scoring systems can help in differentiation of stroke in areas with limited availability of CT scan facility. These scoring systems are based on simple bedside interpretation of clinical findings; it can be used as a diagnostic tool in differentiating stroke in rural and remote hospitals.

Even when the CT brain facility is available, many people are not affordable to utilise the facility. Differentiation of stroke helps in opting for relevant therapeutic options like thrombolysis, anticoagulation, carotid end-arterectomy etc. However in developing countries about 70% of population has poor access to CT brain. Timely diagnosis is especially important in ischemic stroke because early thrombolytic therapy and antiplatelet drugs improves the functional outcome, reduces the morbidity and recurrent strokes. The present study was carried out in 100 consecutive new stroke patients admitted to Coimbatore medical College Hospital, Coimbatore. Those patients who were clinically diagnosed to have stroke were included in the study. The Allen score was calculated for all the stroke patients after 24 hours using calculator. Greek stroke scores were calculated for all the stroke patients at the time of admission.

CT scan brain was taken for all the stroke patients. The stroke scores are computed and compared with imaging studies, with an aim to find the accuracy of Allen score and Greek score in differentiation of stroke subtypes.

SENSITIVITY OF ALLEN SCORE FOR INFARCT:

The sensitivity of Allen stroke score for infarction in the present study is 64.62%. In a study by Akhtar et al, the sensitivity for infarction is 71%. In Raghuram et al study, the sensitivity of Allen score for infarction is 95%. In the present study shows the sensitivity of Allen stroke score is lower compared to other validated studies.

Table 37- comparison of sensitivity of present study other validation studies on Allen stroke score for infarct

STUDY	PLACE OF STUDY	SENSITIVITY
Akhtar et al	Peshawar	71%
Raghuram et al	South India	94%
Present study	Coimbatore	64.62%

SPECIFICITY OF ALLEN SCORE FOR INFARCT:

The specificity of Allen stroke score for infarction in the present study is 60%. In a study by Akhtar et al, [25] the specificity for infarction is 80%. In Raghuram et al [38]study, the specificity of Allen score for infarction is 80%. In the present study shows the specificity of Allen stroke score 60 %, which is contrary to other validated studies.

Table 38- comparison of Specificity of present study other validation studies on Allen stroke score for infarct

STUDY	PLACE OF STUDY	SPECIFICITY
Akhtar et al	Peshawar	80%
Raghuram et al	South India	80%
Present study	Coimbatore	60%

Positive predictive values of Allen score for infarction:

The Positive predictive value of Allen Score in the present study for infarction is 75%. In Akhtar et al [25] study, the positive predictive value in the diagnosis of infarction by Allen score is 89%. In the study conducted by Raghuram et al, [38] the positive predictive value in the diagnosis of infarct by Allen score was 92%. In Rajouria et al [37] study the positive predictive value is 70% which is comparable to our study.

Table 39- Comparison of positive predictive value of Allen score on infarct with other validation studies

STUDY	POSITIVE PREDICTIVE VALUE (%)
Akhthar et al	89
Raghuram et al	92
Rajouria et al	70
Present study	75

SENSITIVITY OF ALLEN SCORE FOR HAEMORRHAGE:

The sensitivity of Allen stroke score for Haemorrhage in the present study is 38.71%. In a study by Akhtar et al, [25] the sensitivity for Haemorrhage is 38%. In Raghuram et al[38] study, the sensitivity of Allen score for Haemorrhage is 80%. In Salawi et al[36] study, the sensitivity for haemorrhage is 64%.

In Nouira et al[41] study, the sensitivity of haemorrhage is 55%. In the present study shows the sensitivity of Allen stroke score is lower compared to other validated studies.

Table 40 - comparison of sensitivity of present study other validation studies on Allen stroke score for Haemorrhage

STUDY	PLACE OF STUDY	SENSITIVITY (%)
Akhtar et all	Peshawar	38
Raghuram et all	South India	80
Salawi et al	Nigeria	64
Nouira et al	America	55
Present study	Coimbatore	38.71

SPECIFICITY OF ALLEN SCORE FOR HAEMORRHAGE:

The Specificity of Allen stroke score for Haemorrhage in the present study is 95.65%. In a study by Akhtar et al, [25] the Specificity for Haemorrhage is 91%. In Raghuram et al [38] study, Specificity of Allen score for Haemorrhage is 94%. In Salawi et al[36] study, the Specificity for haemorrhage is 48%.

In Nouira et al [41]study, Specificity of haemorrhage is 70%. In the present study shows the Specificity of Allen stroke score is comparable with other validated studies.

Table 41- comparison of Specificity of present study with other validation studies on Allen stroke score for Haemorrhage

STUDY	PLACE OF STUDY	SPECIFICITY (%)
Akhtar et all	Peshawar	91
Raghuram et all	South India	94
Salawi et al	Nigeria	48
Nouira et al	America	70
Present study	Coimbatore	95.65

POSITIVE PREDICTIVE VALUE OF ALLEN SCORE FOR HAEMORRHAGE:

The Positive predictive value of Allen stroke score for Haemorrhage in the present study is 80%. In a study by Akhtar et al,[25] the Positive predictive value for Haemorrhage is 66%. In Raghuram et al [38] study, Positive predictive value of Allen score for Haemorrhage is 84%. In Salawi et al [36] study, the Positive predictive value for haemorrhage is 40%. In Nouira et al [41] study, Positive predictive value of haemorrhage is 70%. In the present study shows the Positive predictive value of Allen stroke score is comparable with other validated studies.

Table 42- comparison of Positive predictive value of present study with other validation studies on Allen stroke score for Haemorrhage

STUDY	PLACE OF STUDY	SPECIFICITY (%)
Akhtar et all	Peshawar	66
Raghuram et all	South India	84
Salawi et al	Nigeria	40
Nouira et al	America	70
Present study	Coimbatore	80

STATISTICAL ANALYSIS OF GREEK STROKE SCORE IN DIAGNOSIS OF INFARCT:

The sensitivity, specificity, positive predictive value and negative predictive value of Greek score for diagnosing infarct in our study is 73.85%, 68.57%, 81.36% and 58.54% respectively.

Table 43- statistical analysis of present study with other validation studies on Greek score for Infarct.

STUDY	PLACE	SENSITIVITY (%)	SPECIFICITY (%)
Berhe et al	Ethiopia	59.5	65
Clifford et al	Kenya	70	79
Present study	Coimbatore	73.85	68.57

In Berhe et al study, [39] the sensitivity and specificity of Greek score for diagnosing Infarction is 59.5% and 65% respectively. Clifford study [26] showed the sensitivity and specificity of Greek stroke for diagnosing Infarction is 70% and 79% respectively.

STATISTICAL ANALYSIS OF GREEK STROKE SCORE IN DIAGNOSIS OF HAEMORRHAGE:

The sensitivity, specificity, positive predictive value and negative predictive value of Greek score for diagnosing Haemorrhage in our study is 25.81%, 91.1%, 80 and 74.44% respectively. In Rajouria et al study, [37] sensitivity is 85%, specificity is 73%, and positive predictive value is 69%. Clifford study [26] showed the sensitivity and specificity of Greek stroke for diagnosing haemorrhage is 54% and 89% respectively.

Table 44- statistical analysis of present study with other validation studies on Greek score for Haemorrhage.

STUDY	PLACE	SENSITIVITY (%)	SPECIFICITY (%)
Berhe et al	Ethiopia	78.8	89.3
Greek study	Athens	99	99
Clifford et al	Kenya	54	89
Rajouria et al	Kathmandu	85	73
Present study	Coimbatore	25.81	91.1

In Berhe et al study, [39] the sensitivity and specificity of Greek score for diagnosing haemorrhage is 78.8% and 89.3% respectively. The original Greek study [30] from Athens showed sensitivity of 99%, specificity of 99%, positive predictive value of 97%, negative predictive value of 97%.

All the scoring systems are calculated based on clinical symptoms. Allen score showed definite results in 68 patients and equivocal in 28 patients, hence the applicability rate of Allen score is 68%. Greek score showed definite results in 59 patients and equivocal in 9 patients, hence the applicability rate is 59%. In Allen score more weightage of points are given to the apoplectic onset i.e headache, vomiting, neck stiffness and level of consciousness on admission & at 24 hours. Level of consciousness is given more points in Greek stroke score too. In our Study, patients with massive infarct in CT scan brain were found to have haemorrhage by the stroke scores. It may be due to points given for apoplectic onset which is in more favour of haemorrhage. Variables used in the study are not given with proper definitions. The massive infarct and haemorrhage can cause cerebral oedema which leads to midline shift; compression of brain stem, alteration in conscious level makes the calculation of stroke score less useful.

There is need for alteration in the variables used in the scoring systems. Hypertension is one of the most important risk factor for both infarct as well as haemorrhage, but in Allen and Greek scores, hypertension has more association with infarction and haemorrhage.

When CT scan is not available and the main concern for the treating physician is to rule out haemorrhage to start antithrombotic therapy. Therefore a test with highest specificity and negative predictive value for haemorrhage is essential. In present study Allen score has the highest specificity for haemorrhage than Greek score. Greek score has the highest negative predictive value for haemorrhage than Allen score.

CONCLUSION

The Greek Stroke score and Allen Score are easy and faster to calculate. But they are prone to mathematical errors. Even though the sensitivity, specificity and negative predictive values to detect haemorrhage is much better in Greek Stroke Score than Allen's score, the sensitivity and specificity are not comparable to CT Brain .The diagnostic accuracy of Allen and Greek score for infarct is 63% and 72% respectively. The diagnostic accuracy of Allen score & Greek score for haemorrhage is 78% & 75% respectively. Further larger studies using different populations are needed before using stroke scores as a screening tool in the diagnosis of stroke. Stroke scoring systems are still not accurate to replace CT scan as investigation of choice even when the facility is not feasible.

BIBLIOGRAPHY

1. www.hopkinsmedicine.org/healthlibrary
2. J.P Mohr, Dennis-Choi- epidemiology of stroke 2:13, J.P Mohr, Dennis-Choi- classification of stroke chapter-4
3. Merritt's neurology chapter 35:217-222
4. Fundamentals of neurology 2004 chapter 6: diseases of brain and meninges page no. 147 - 157
5. Fundamentals of neurology 2004 chapter 34: 660
6. ncbi.nim.nih.gov/books chapter 5- control of cerebral blood flow
7. Middle cerebral artery- review article; radiopaedia.org
8. Classification of subtype of acute ischemic stroke- TOAST study 1993; 24:35-41
9. Stroke subtype classification by geometrical descriptors of lesion shape; PLos 2017; 12(12):e0185063
10. Classification of intracerebral haemorrhages – European neurological review, 2014;9(2):129-35
11. Update in intracerebral haemorrhage- neurohospitalist. 2011 Jul; 1(3):148-159

12. Risk factors of stroke- Philip a wolf – stroke 16(3), 359- 360, 1985.

13. www.stroke association.org – stroke risk factors.

14. Cardiovascular risk factors for acute stroke; risk profiles, 2015 May 16:3(5): 418-429.

15. Therapeutic advances in cardiovascular disease- stroke prevention: modifying risk factors. 2008 Aug; 2 (4):287-303.

16. www.ncbi.nlm.gov/pmc/articles 2008 May 8.doi 10.1186/1752

17. www.radiopaedia.org

18. The role of neuroimaging in acute stroke –www.annalsofian.org review article- 2008, volume 11-page 12-23.

19. www.uptodate.com – review literature Mar 13, 2018

20. European neurology – imaging of acute ischemic stroke 2014; 72:309-316

21. Neurosurgery focus 36(1):E3, 2014- the role of imaging in acute ischemic stroke. Neurosurgery focus vol.36, issue 1; January 2014- imaging of high risk carotid artery plaques. Haemorrhagic stroke treatment and management – Medscape- Jan 22, 2017 Fundamentals of neurology- Heinrich Mattle & Marco Mumenthaler Chapter 6: diseases of brain & meninges, 6.5; page number: 148 -155.

22. Dalsgaard Midson T. Survey of 1000 cases of apoplexy cerebri. Acta Psychiatric neurological Scandinavia 1956; 30:169.

23. Bamford J, Sandercock P, Dennis M et al. A prospective study of Acute cerebrovascular disease in the community. Journal of Neurol. Neurosurg. Psychiatry 1988; 51:1373-80

24. Recovery of motor function after stroke 1988; 19:1497-500. Bonita R, Beaglehole. Allen CMC - Clinical diagnosis of acute stroke syndrome. Quarterly Journal of Medicine 1983; 52: 515.

25. Poungvarin N, Viriyavejakul A, Komontri C. Siriraj stroke score And validation study to distinguish supratentorial intracerebral Haemorrhage from infarction. BMJ. 1991; 302:1565-67

26. Efstathiou SP, Tsioulos DI, Zacharos ID, Tsiakou AG, Mitromaras AG, Mastorantonakis SE, et al. A new classification tool for Clinical differentiation between haemorrhagic and ischemic stroke. Journal of internal medicine. 2002; 252(2):121-9.

27. Soman A, Joshi Shashank R, Travade S et al. Greek stroke score,Siriraj score and Allen score in clinical diagnosis of intra-cerebral Haemorrhage and infarct: validation and comparison study. Indian Journal -Med Sci. 2004; 58:417-422.

28. Huang, Wang PY, Chang MC, Chia LG, Yang DY, Wu TC. Allen score in clinical diagnosis of intracerebral haemorrhage. Zhonghua Yi Xue Za Zhi - 1994; 45(6):417-411.

29. Sandercock PA, Allen CM, Corston RN, Harrison MJ, Warlow CP. Clinical diagnosis of intracerebral haemorrhage using Guy's Hospital score. Br Med J (clin Res Ed) – 1985; 291(6510):1675-7.

30. Salawu F, Umar I, Danburam A. Comparison of two hospital stroke score with computerized tomography in ascertaining stroke types Nigerians. Annals of African Medicine. 2009; 8:1:14-18.

31. 35.Rajouria AD, Rana KJ, Karki L, Gaire D, Pokheral A – National academy of medical sciences volume 12,number 1, Jan – Jul 2012, 13-17.

32. Pavan Manibettu Raghuram, Mallanagouda Shivanagoudabiradar, Jayakumar Jeganathan - Comparison of Siriraj stroke and the Guy's hospital score in South India. DOI: JCDR/2012/4406:0000; 850 – 853.

33. T.Berhe, G.Zenebe, Y Melkamu – Application of Greek Stroke Score in Ethiopia - A Validation Study; the Internet Journal of Neurology, Volume 11, Number 1.

34. A.Ozarean, S.Bicakci, R.Burgut, Y.Sarica, H. Bozdemir – Accuracy of Bedside Versus Allen and Siriraj Stroke Score in Turkish Patients; European Journal of Neurology / volume 13, issue 6.

35. Semir Nouira, Riadh Boukef , Wahid Bouida, Sodani Marghli – Accuracy of Two Scores in the diagnosis of Stroke Subtype in a Multicentre Cohort Study - Annals of Emergency Medicine; August 19,2008.

APPENDIX I- PROFORMA

COMPARISON OF ALLEN STROKE SCORE AND GREEK STROKE SCORE WITH CT BRAIN IN CLINICAL DIAGNOSIS OF ACUTE STROKE

NAME: AGE/SEX:

I.P.NO: UNIT:

OCCUPATION: ADDRESS:

COMPLAINTS:

PRESENTING SYMPTOMS:

- DURATION
- MODE OF ONSET
- TIME OF ONSET

APOPLECTIC SYMPTOMS:

- HEADACHE/VOMITING/LOC
- HISTORY SUGGESTIVE OF CRANIAL NERVE INVOLVEMENT
- HISTORY OF SENSORY DISTURBANCES
- H/O BOWEL/BLADDER DISTURBANCES
- H/O FEVER, BLURRING OF VISION, SEIZURES
- H/O CHEST PAIN, PALPITATION, LIMB PAIN

PAST HISTORY:

- HT /DM/ CAD/RHD/ ANY OTHER HEART DISEASE /TIA/CVA

PERSONAL HISTORY:

- DIET:
- SMOKING:
- ALCHOCOL:

FAMILY HISTORY:

TREATMENT HISTORY:

- H/O INTAKE OF ANTIPLATELET DRUGS: YES/NO x
- IF YES INDICATION:

EXAMINATION

GENERAL EXAMINATION

- CONCIOUS LEVEL AT ADMISSION 24 HOURS AFTER ADMISSION ALERT/DROWSY/UNCONSCIOUS
- ORIENTATION
- PALLOR/ICTERUS
- CYANOSIS/CLUBBING
- PEDAL EDEMA
- NEUROCUTANEOUS MARKERS

VITAL SIGNS

- PULSE:
- BP

AT ADMISSION:

24 HOURS AFTER ADMISSION:

- RESPIRATORY RATE:
- TEMPERATURE:

SYSTEMIC EXAMINATION:

CNS:

- HIGHER MENTAL FUNCTIONS:
- CRANIAL NERVES EXAMINATION
- MOTOR SYSTEM:
- SENSORY SYSTEM:
- AUTONOMIC NERVOUS SYSTEM:
- CEREBELLAR FUNCTIONS:
- SPINE AND CRANIUM:
- MENINGEAL SIGNS:

CARDIOVASCULAR SYSTEM:

RESPIRATORY SYSTEM:

ABDOMEN:

INVESTIGATIONS:

- COMPLETE BLOOD COUNT
- CT SCAN BRAIN

CALCULATION OF ALLEN AND GREEK SCORE:

ALLEN SCORE & GREEK SCORE WITH CT SCAN:

CT BRAIN REPORT:

APPENDIX II

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«è£¬õ Üó² ñ¼^¶õ, è™ÖK ñ¼^¶õñ¬ùJ™ ñ¼. r.nkdfh î¬ô¬ñJ™ ï¬ìªðÁ‹ Þ‰î ÝŒM™ âù¶ º¿àì™ ñŸÁ‹ Þó^îŠ ðK«ê£î¬ù ªêŒ¶ ªè£œ÷ º¿ ñù¶ì¡ ê‹ñF,A«ø¡. â¡¬ùŠ ðŸPò Mõóƒè¬÷ ð£¶è£Š¹ì¡ Þ‰î ÝŒM™ ªõOJì Ý†«êð¬í Þ™¬ô â¡Á ªîKM^¶, ªè£œA«ø¡. ï£¡ â‰î «ïó^F½‹ ÝŒM™ Þ¼‰¶ Mô,A, ªè£œÀ‹ àK¬ñ à‡´ â¡Á ÜP«õ¡.

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APPENDIX III - KEY TO MASTERCHART

1	S.NO – SERIAL NUMBER	
2	AGE	
3	SEX	M – MALE F – FEMALE
4	COMPLAINTS	1-RIGHT HEMIPLEGIA 2- RIGHT HEMIPARESIS 3 – LEFT HEMIPLEGIA 4 – LEFT HEMIPARESIS
5	VOM - VOMITING	0 – ABSENT 1- PRESENT
6	HA - HEADACHE	0 - ABSENT 1 - PRESENT
7	HT – HYPERTENSION	0 - ABSENT 1 – PRESENT
8	AAM – ATHEROMA MARKERS	0 - ABSENT 1 – PRESENT
9	HD – HEART DISEASES	0 - ABSENT 1 – PRESENT
10	TIA/STR – PAST HISTORY OF STROKE	0 - ABSENT 1 – PRESENT
11	SMO – SMOKING	0 - ABSENT 1 – PRESENT
12	ALC – ALCOHOL	0 - ABSENT 1 – PRESENT
13	APO-ON – APOPLECTIC ONSET	0 - ABSENT 1 – PRESENT
14	C.ADMN - CONDITION AT THE TIME OF ADMISSION	0 – ALERT 1 – DROWSY 2 - UNCONSCIOUS
15	C.24 – CONDITION AFTER 24 HOURS OF ADMISSION	0 – ALERT 1 – DROWSY 2 – UNCONSCCIOUS
16	DBP ADMN –	

	DIASTOLIC BLOOD PRESSURE AT THE TIME OF ADMISSION	
17	TC – TOTAL COUNT	0 – <12000 1 - > 12000
18	PL – PLANTAR RESPONSE	0 – BILATERAL FLEXOR 1 – UNILATERAL EXTENSOR 2 – BILATERAL EXTENSOR
19	ASS – ALLEN STROKE SCORE	<4 – INFARCT 5-24 – EQUIVOCAL >24 – HEMORRHAGE
20	GSS – GREEK STROKE SCORE	LESS THAN OR EQUAL TO 3 – INFARCT 3-12 – EQUIVOCAL >12 - HEMORRHAGE
21	CT – CT BRAIN : HEGE – HEMORRHAGE CVT – CEREBRAL VENOUS THROMBOSIS	

APPENDIX IV - MASTER CHART

S.NO	AGE	SEX	COMPLAINTS	VOM	HA	HT	AAM	HD	TIA/ST	SMO	ALC	APO-ON	C.ADMN	C.24	DBP ADMN	PLT	TC	ASS	GSS	CT
1	65	M	1	0	0	1	0	0	0	1	1	1	2	1	90	1	0	5.9	3	INFARCT
2	55	F	3	1	0	0	0	0	0	0	0	1	1	0	100	1	0	3	7	HEGE
3	48	F	2	0	0	0	1	0	0	0	0	1	1	0	100	1	1	0.7	2	INFARCT
4	46	M	0	1	1	0	0	0	0	1	1	1	2	1	90	0	0	33.6	7	CVT
5	52	F	1	0	0	1	0	0	0	0	0	0	0	0	110	1	0	14.6	0	INFARCT
6	36	F	2	1	1	0	0	0	0	1	0	1	0	1	90	0	1	31.9	14	TUBERCULOMA
7	56	M	1	0	0	1	1	0	0	0	1	0	1	0	100	0	0	-3.4	0	INFARCT
8	53	M	4	0	1	1	0	0	0	1	1	0	2	1	100	0	0	20.2	3	INFARCT
9	38	F	3	1	1	0	0	0	0	0	0	1	0	0	70	0	1	33.8	7	HEGE
10	63	M	2	0	0	1	1	0	0	2	1	0	0	2	90	0	0	17.3	0	INFARCT
11	59	F	3	0	0	1	0	1	0	0	0	0	1	0	80	0	0	-8.3	4	INFARCT
12	48	F	4	0	1	1	1	1	0	0	0	0	2	0	60	0	0	-7.3	0	INFARCT
13	52	M	1	0	0	1	0	0	0	1	1	0	0	1	90	1	0	2.6	4	INFARCT
14	73	F	2	0	0	1	0	0	1	0	1	0	0	0	90	2	0	-1	0	INFARCT
15	68	M	3	0	0	1	1	0	0	0	0	0	2	0	90	2	0	2	0	HEGE
16	75	F	4	1	1	0	1	0	0	2	0	1	1	2	100	2	1	20.2	16	HEGE
17	38	M	3	0	1	1	0	0	0	0	1	0	0	0	80	2	1	7.4	3	INFARCT
18	65	F	2	0	0	1	1	0	0	0	0	0	0	0	90	0	0	-3.4	0	INFARCT
19	39	M	2	1	0	0	0	1	0	1	0	0	2	1	90	0	0	7.2	4	HEGE
20	58	F	4	0	0	1	1	0	0	1	0	0	0	1	80	1	0	0.5	0	INFARCT
21	72	F	1	1	0	1	0	0	0	0	0	1	1	0	110	2	1	38.4	14	HEGE
22	56	M	4	0	0	1	0	0	0	2	1	0	0	2	90	2	0	20.4	6	INFARCT
23	40	M	2	0	0	1	0	0	0	0	1	0	2	0	70	0	0	-4.8	0	INFARCT
24	60	F	3	0	1	1	1	0	0	1	0	0	0	1	80	2	0	0.5	0	HEGE
25	74	M	0	0	0	1	1	0	0	0	0	0	1	0	90	0	1	-4.3	4	INFARCT
26	49	F	1	0	1	1	0	0	1	1	1	0	0	1	80	0	0	-2.5	0	INFARCT
27	57	M	4	1	0	0	0	1	0	2	1	0	2	2	100	2	0	21.8	4	HEGE
28	72	M	3	0	0	1	1	0	0	0	1	0	0	0	90	0	0	0.3	0	INFARCT

29	50	F	2	1	1	0	0	1	0	0	0	1	1	0	110	0	0	25	4	HEGE
30	62	M	2	0	0	1	1	0	0	0	1	0	0	0	80	2	0	-0.3	0	INFARCT
31	47	M	1	0	0	0	0	0	0	1	0	0	0	1	90	0	0	3	0	INFARCT
32	60	F	3	1	1	1	1	0	0	0	0	0	1	0	70	2	1	19.9	14	INFARCT
33	52	F	2	0	0	0	0	0	0	2	0	0	0	2	90	1	0	27	4	HEGE
34	76	M	4	0	0	1	0	0	0	0	1	0	2	0	80	0	0	-1.5	0	INFARCT
35	82	F	1	0	0	0	0	0	0	0	0	0	0	0	80	1	0	1	0	INFARCT
36	45	M	0	1	1	0	0	0	0	0	1	1	1	0	90	0	1	24.6	14	HEGE
37	65	F	2	1	1	1	0	0	0	1	0	0	0	1	100	2	0	25	0	HEGE
38	58	M	2	0	1	1	1	0	0	2	1	0	1	2	90	0	0	0.8	0	INFARCT
39	46	F	2	0	0	1	1	0	1	0	0	0	0	0	90	1	1	-12.6	4	INFARCT
40	50	M	1	0	0	1	0	0	0	0	0	0	2	0	80	0	0	-3.1	0	INFARCT
41	47	M	4	0	0	0	1	1	0	2	0	0	0	2	80	0	0	7.8	13	HEGE
42	62	F	1	0	0	1	0	0	0	0	0	0	0	0	100	2	0	-2.3	0	INFARCT
43	59	M	3	0	0	1	0	0	0	1	1	0	1	1	90	0	0	5.9	0	INFARCT
44	40	M	2	1	1	0	0	1	0	0	1	1	0	0	90	0	0	2	4	INFARCT
45	52	F	4	0	1	1	0	0	1	0	0	0	0	0	90	0	0	34	4	HEGE
46	65	M	0	0	0	1	1	0	0	0	1	0	1	0	80	1	0	1	0	INFARCT
47	56	F	2	0	0	1	0	0	0	1	1	0	2	1	60	0	1	1.5	4	INFARCT
48	45	M	2	0	0	1	0	0	0	0	1	0	0	0	100	2	0	0.3	0	INFARCT
49	36	M	3	1	0	0	0	0	0	2	1	0	0	2	90	0	0	17.3	4	CVT
50	44	M	2	0	1	1	0	0	1	0	0	0	1	0	70	1	0	-8.5	0	INFARCT
51	52	F	1	1	0	1	0	0	0	0	0	1	0	0	90	0	0	6.2	0	INFARCT
52	62	F	4	0	0	1	0	0	0	0	0	0	0	0	110	2	0	8.6	8	HEGE
53	47	M	4	0	1	0	0	1	0	1	0	0	1	1	70	0	0	0	3	INFARCT
54	64	M	4	0	0	1	0	0	0	1	1	0	0	1	90	0	0	-2.3	4	INFARCT
55	50	M	2	0	0	1	0	0	0	0	1	0	2	0	100	2	0	29	0	INFARCT
56	56	M	1	1	1	1	1	0	0	0	1	1	0	0	80	0	1	3	3	HEGE
57	32	M	1	0	0	0	0	0	0	2	1	0	1	2	70	0	0	5	4	INFARCT
58	60	M	1	0	1	1	1	0	0	0	1	0	0	0	90	2	0	9	0	INFARCT
59	42	M	1	0	0	1	0	0	0	0	1	0	0	0	80	1	0	11	0	HEGE

60	56	M	3	0	0	0	0	1	0	1	1	0	1	1	90	0	0	-8.5	0	INFARCT
61	46	M	2	1	0	1	0	0	0	0	1	1	0	0	90	1	0	16	4	INFARCT
62	61	F	3	0	0	1	0	0	1	2	0	0	2	2	90	0	0	-3.4	0	HEGE
63	42	M	2	0	0	1	0	0	0	0	1	0	0	0	100	2	0	10	0	INFARCT
64	40	M	3	0	0	0	0	0	0	0	1	0	0	0	90	0	0	24.9	14	HEGE
65	56	M	4	0	0	1	1	0	0	2	0	0	0	2	70	0	0	-7.3	0	HEGE
66	64	F	1	0	0	0	0	0	0	0	0	0	1	0	60	0	0	19	10	INFARCT
67	48	M	3	0	0	1	0	0	0	0	1	0	0	0	90	2	0	2.6	0	INFARCT
68	54	F	2	0	0	1	0	0	0	1	0	0	0	1	90	1	0	12	0	INFARCT
69	66	F	1	0	0	1	0	0	0	2	0	0	2	2	90	0	0	0	10	HEGE
70	46	M	3	0	0	0	0	0	0	0	1	0	0	0	70	0	0	-1	0	INFARCT
71	55	F	4	0	1	1	1	1	0	0	1	1	0	0	100	2	1	0.3	4	INFARCT
72	47	M	1	0	0	1	0	0	0	1	0	0	1	1	90	0	0	16	0	INFARCT
73	58	F	2	0	0	1	0	0	1	0	0	0	0	0	90	0	0	0.5	4	INFARCT
74	50	M	0	0	0	0	0	0	0	0	0	0	0	0	90	0	0	0	7	HEGE
75	34	M	1	0	1	1	0	0	0	2	0	0	0	2	120	2	0	-4.3	0	INFARCT
76	55	M	3	1	0	1	0	1	0	0	1	1	1	0	90	1	0	0.5	0	GLIOMA
77	46	M	2	0	0	0	0	0	0	0	1	0	2	0	90	0	0	25	0	HEGE
78	66	F	4	0	0	1	0	0	0	1	0	0	0	1	110	2	0	3	4	INFARCT
79	53	M	4	0	0	1	0	0	0	0	1	0	0	0	90	0	0	6.8	0	INFARCT
80	38	M	1	0	1	1	0	0	0	0	1	1	0	0	80	0	1	-2.5	10	HEGE
81	46	M	2	0	0	0	0	0	0	0	1	0	1	0	90	1	0	9	0	INFARCT
82	53	M	3	0	0	1	0	0	0	1	1	0	0	1	90	0	0	25	16	HEGE
83	49	M	3	0	0	1	0	0	0	0	1	0	0	0	90	1	0	0.3	0	INFARCT
84	54	M	2	0	1	0	0	0	0	0	1	1	2	0	90	0	0	9	4	INFARCT
85	62	F	4	0	0	1	0	0	0	1	0	0	0	1	100	2	0	29	0	HEGE
86	50	M	3	0	0	1	0	0	0	2	1	0	0	2	90	0	0	11	0	INFARCT
87	54	F	1	0	0	1	1	0	0	0	0	0	1	0	140	1	0	-12.6	0	HEGE
88	35	M	1	0	0	0	1	1	0	0	1	0	0	0	90	2	0	-7.3	14	HEGE
89	58	M	2	0	1	1	0	0	0	1	1	0	0	1	90	1	1	2.6	0	INFARCT
90	46	M	4	0	0	1	0	0	1	0	1	0	2	0	140	2	0	-1	3	INFARCT

91	60	M	2	0	0	1	1	0	0	0	1	0	0	0	90	0	0	9	0	INFARCT
92	52	M	4	0	1	1	0	0	0	1	1	0	1	1	90	1	0	0	7	INFARCT
93	48	M	1	1	0	1	0	0	0	0	1	1	0	0	80	0	0	-2	0	INFARCT
94	56	F	1	0	0	1	0	1	0	2	0	0	0	2	100	1	0	0	16	HEGE
95	61	M	4	0	0	1	0	0	0	0	0	0	2	0	90	1	1	-3.2	0	INFARCT
96	55	M	2	0	0	0	0	0	0	0	1	0	0	0	90	0	0	0.3	0	INFARCT
97	40	M	2	0	0	1	0	0	0	1	0	0	0	1	80	1	0	-3.4	4	HEGE
98	52	M	4	0	0	1	0	0	0	0	1	0	1	0	90	1	0	4	0	INFARCT
99	45	M	1	0	0	1	0	0	0	0	0	0	0	0	90	0	0	4.2	0	INFARCT
100	58	M	2	0	0	1	0	0	0	1	1	0	2	1	100	2	0	26.2	4	HEGE